TARGETED CHROMOSOMAL GENOMIC ALTERATIONS WITH MODIFIED SINGLE STRANDED OLIGONUCLEOTIDES

This application claims benefit from United States Provisional Application No. 60/192,176, filed May 27, 2000; United States Provisional Application No. 60/192,179, filed May 27, 2000; United States Provisional Application No. 60/208,538, filed June 1, 2000; and United States Provisional Application No. 60/244,989, filed October 30, 2000.

Field Of The Invention

The technical field of the invention is oligonucleotide-directed repair or alteration of genetic information using novel chemically modified oligonucleotides. Such genetic information is preferably from a eukaryotic organism, i.e. a plant, animal or fungus.

Background Of The Invention

A number of methods have been developed specifically to alter the sequence of an isolated DNA in addition to methods to alter directly the genomic information of various plants, fungi and animals, including humans ("gene therapy"). The latter methods generally include the use of viral or plasmid vectors carrying nucleic acid sequences encoding partial or complete portions of a particular protein which is expressed in a cell or tissue to effect the alteration. The expression of the particular protein then results in the desired phenotype. For example, retroviral vectors containing a transgenic DNA sequence allowing for the production of a normal CFTR protein when administered to defective cells are described in U.S. Patent 5,240,846. Others have developed different "gene therapy vectors" which include, for example, portions of adenovirus (Ad) or adeno-associated virus (AAV), or other viruses. The virus portions used are often long terminal repeat sequences which are added to the ends of a transgene of choice along with other necessary control sequences which allow expression of the transgene. See U.S. Patents 5,700,470 and 5,139,941. Similar methods have been developed for use in plants. See, for example, U.S. Patent 4,459,355 which describes a method for transforming plants with a DNA vector and U.S. Patent 5,188,642 which describes cloning or expression vectors containing a transgenic DNA sequence which when expressed in plants confers resistance to the herbicide glyphosate. The use of such transgene vectors in any eukaryotic organism adds one or more exogenous copies of a gene, which

gene may be foreign to the host, in a usually random fashion at one or more integration sites of the organism's genome at some frequency. The gene which was originally present in the genome, which may be a normal allelic variant, mutated, defective, and/or functional, is retained in the genome of the host.

TARGETED CHROMOSOMAL GENOMIC ALTERATIONS WITH MODIFIED SINGLE STRANDED OLIGONUCLEOTIDES

Field Of The Invention

The technical field of the invention is oligonucleotide-directed repair or alteration of genetic information using novel chemically modified oligonucleotides. Such genetic information is preferably from a eukaryotic organism, i.e. a plant, animal or fungus.

Background Of The Invention

A number of methods have been developed specifically to alter the sequence of an isolated DNA in addition to methods to alter directly the genomic information of various plants, fungi and animals, including humans ("gene therapy"). The latter methods generally include the use of viral or plasmid vectors carrying nucleic acid sequences encoding partial or complete portions of a particular protein which is expressed in a cell or tissue to effect the alteration. The expression of the particular protein then results in the desired phenotype. For example, retroviral vectors containing a transgenic DNA sequence allowing for the production of a normal CFTR protein when administered to defective cells are described in U.S. Patent 5,240,846. Others have developed different "gene therapy vectors" which include, for example, portions of adenovirus (Ad) or adeno-associated virus (AAV), or other viruses. The virus portions used are often long terminal repeat sequences which are added to the ends of a transgene of choice along with other necessary control sequences which allow expression of the transgene. See U.S. Patents 5,700,470 and 5,139,941. Similar methods have been developed for use in plants. See, for example, U.S. Patent 4,459,355 which describes a method for transforming plants with a DNA vector and U.S. Patent 5,188,642 which describes cloning or expression vectors containing a transgenic DNA sequence which when expressed in plants confers resistance to the herbicide glyphosate. The use of such transgene vectors in any eukaryotic organism adds one or more exogenous copies of a gene, which gene may be foreign to the host, in a usually random fashion at one or more integration sites of the organism's genome at some frequency. The gene which was originally present in the genome, which may be a normal allelic variant, mutated, defective, and/or functional, is retained in the genome of the host.

These methods of gene correction are problematic in that complications which can compromise the health of the recipient, or even lead to death, may result. One such problem is that insertion of exogenous nucleic acid at random location(s) in the genome can have deleterious effects. Another problem with such systems includes the addition of unnecessary and unwanted genetic material to the genome of the recipient, including, for example, viral or other vector remnants, control sequences required to allow production of the transgene protein, and reporter genes or resistance markers. Such remnants and added sequences may have presently unrecognized consequences, for example, involving genetic rearrangements of the recipient genomes. Other problems associated with these types of traditional gene therapy methods include autoimmune suppression of cells expressing an inserted gene due to the presence of foreign antigens. Concerns have also been raised with consumption, especially by humans, of plants containing exogenous genetic material.

More recently, simpler systems involving poly- or oligo- nucleotides have been described for use in the alteration of genomic DNA. These chimeric RNA-DNA oligonucleotides, requiring contiguous RNA and DNA bases in a double-stranded molecule folded by complementarity into a double hairpin conformation, have been shown to effect single basepair or frameshift alterations, for example, for mutation or repair of plant or animal genomes. See, for example, WO 99/07865 and U.S. Patent 5,565,350. In the chimeric RNA-DNA oligonucleotide, an uninterrupted stretch of DNA bases within the molecule is required for sequence alteration of the targeted genome while the obligate RNA residues are involved in complex stability. Due to the length, backbone composition, and structural configuration of these chimeric RNA-DNA molecules, they are expensive to synthesize and difficult to purify. Moreover, if the RNA-containing strand of the chimeric RNA-DNA oligonucleotide is designed so as to direct gene conversion, a series of mutagenic reactions resulting in nonspecific base alteration can result. Such a result compromises the utility of such a molecule in methods designed to alter the genomes of plants and animals, including in human gene therapy applications.

Alternatively, other oligo- or poly- nucleotides have been used which require a triplex forming, usually polypurine or polypyrimidine, structural domain which binds to a DNA helical duplex through Hoogsteen interactions between the major groove of the DNA duplex and the oligonucleotide. Such oligonucleotides may have an additional DNA reactive moiety, such as psoralen, covalently linked to the oligonucleotide. These reactive moieties function as effective intercalation agents, stabilize the formation of a triplex and can be mutagenic. Such agents may be required in order to stabilize the triplex forming domain of the oligonucleotide with the DNA double helix if the Hoogsteen interactions from the oligonucleotide/target base composition are insufficient. See, e.g., U.S. Patent 5,422,251. The utility of

these oligonucleotides for directing gene conversion is compromised by a high frequency of nonspecific base changes.

In more recent work, the domain for altering a genome is linked or tethered to the triplex forming domain of the bi-functional oligonucleotide, adding an additional linking or tethering functional domain to the oligonucleotide. See, e.g., Culver et al., Nature Biotechnology 17: 989-93 (1999). Such chimeric or triplex forming molecules have distinct structural requirements for each of the different domains of the complete poly- or oligo-nucleotide in order to effect the desired genomic alteration in either episomal or chromosomal targets.

Other genes, e.g. CFTR, have been targeted by homologous recombination using duplex fragments having several hundred basepairs. See, e.g., Kunzelmann et al., Gene Ther. 3:859-867 (1996). Early experiments to mutagenize an antibiotic resistance indicator gene by homologous recombination used an unmodified DNA oligonucleotide with no functional domains other than a region of complementary sequence to the target. See Campbell et al., New Biologist 1: 223-227 (1989). These experiments required large concentrations of the oligonucleotide, exhibited a very low frequency of episomal modification of a targeted exogenous plasmid gene not normally found in the cell and have not been reproduced. However, as shown in the examples herein, we have observed that an unmodified DNA oligonucleotide can convert a base at low frequency which is detectable using the assay systems described herein.

Artificial chromosomes can be useful for the screening purposed identified herein. These molecules are man-made linear or circular DNA molecules constructed from essential cis-acting DNA sequence elements that are responsible for the proper replication and partitioning of natural chromosomes (Murray et al., 1983). The essential elements are: (1) Autonomous Replication Sequences (ARS), (2) Centromeres, and (3) Telomeres.

Yeast artificial chromosomes (YACs) allow large genomic DNA to be modified and used for generating transgenic animals [Burke et al., Science 236:806; Peterson et al., Trends Genet. 13:61 (1997); Choi, et al., Nat. Genet., 4:117-223 (1993), Davies, et al., Biotechnology 11:911-914 (1993), Matsuura, et al., Hum. Mol. Genet., 5:451-459 (1996), Peterson et al., Proc. Natl. Acad. Sci., 93:6605-6609 (1996); and Schedl, et al., Cell, 86:71-82 (1996)]. Other vectors also have been developed for the cloning of large segments of mammalian DNA, including cosmids, and bacteriophage P1 [Sternberg et al., Proc. Natl. Acad. Sci. U.S.A., 87:103-107 (1990)]. YACs have certain advantages over these alternative large capacity cloning vectors [Burke et al., Science, 236:806-812 (1987)]. The

maximum insert size is 35-30 kb for cosmids, and 100 kb for bacteriophage P1, both of which are much smaller than the maximal insert for a YAC.

An alternative to YACs are E. coli based cloning systems based on the E. coli fertility factor that have been developed to construct large genomic DNA insert libraries. They are bacterial artificial chromosomes (BACs) and P-1 derived artificial chromosomes (PACs) [Mejia et al., Genome Res. 7:179-186 (1997); Shizuya et al., Proc. Natl. Acad. Sci. 89:8794-8797 (1992); Ioannou et al., Nat. Genet., 6:84-89 (1994); Hosoda et al., Nucleic Acids Res. 18:3863 (1990)]. BACs are based on the E. coli fertility plasmid (F factor); and PACs are based on the bacteriophage P1. These vectors propagate at a very low copy number (1-2 per cell) enabling genomic inserts up to 300 kb in size to be stably maintained in recombination deficient hosts. Furthermore, the PACs and BACs are circular DNA molecules that are readily isolated from the host genomic background by classical alkaline lysis [Birnboim et al., Nucleic Acids Res. 7:1513-1523 (1979].

Oligonucleotides designed for use in the alteration of genetic information are significantly different from oligonucleotides designed for antisense approaches. For example, antisense oligonucleotides are perfectly complementary to and bind an mRNA strand in order to modify expression of a targeted mRNA and are used at high concentration. As a consequence, they are unable to produce a gene conversion event by either mutagenesis or repair of a defect in the chromosomal DNA of a host genome. Furthermore, the backbone chemical composition used in most oligonucleotides designed for use in antisense approaches renders them inactive as substrates for homologous pairing or mismatch repair enzymes and the high concentrations of oligonucleotide required for antisense applications can be toxic with some types of nucleotide modifications. In addition, antisense oligonucleotides must be complementary to the mRNA and therefore, may not be complementary to the other DNA strand or to genomic sequences that span the junction between intron sequence and exon sequence.

A need exists for simple, inexpensive oligonucleotides capable of producing targeted alteration of genetic material such as those described herein as well as methods to identify optimal oligonucleotides that accurately and efficiently alter target DNA.

Summary Of The Invention

Novel, modified single-stranded nucleic acid molecules that direct gene alteration in plants, fungi and animals are identified and the efficiency of alteration is analyzed both in vitro using a cell-free extract assay and in vivo using a yeast cell system. The alteration in an oligonucleotide of the invention may comprise an insertion, deletion, substitution, as well as any combination of these. Site

specific alteration of DNA is not only useful for studying function of proteins in vivo, but it is also useful for creating animal models for human disease, and in gene therapy. As described herein, oligonucleotides of the invention target directed specific gene alterations in genomic double-stranded DNA cells. The target DNA can be normal, cellular chromosomal DNA, extrachromosomal DNA present in cells in different forms including, e.g., mammalian artificial chromosomes (MACs), PACs from P-1 vectors, yeast artificial chromosomes (YACs), bacterial artificial chromosomes (BACs), plant artificial chromosomes (PLACs), as well as episomal DNA, including episomal DNA from an exogenous source such as a plasmid or recombinant vector. Many of these artificial chromosome constructs containing human DNA can be obtained from a variety of sources, including, e.g., the Whitehead Institute, and are described, e.g., in Cohen et al., Nature 336:698-701 (1993) and Chumakov, et al., Nature 377:174-297 (1995). The target DNA may be transcriptionally silent or active. In a preferred embodiment, the target DNA to be altered is the non-transcribed strand of a genomic DNA duplex.

The low efficiency of gene alteration obtained using unmodified DNA oligonucleotides is believed to be largely the result of degradation by nucleases present in the reaction mixture or the target cell. Although different modifications are known to have different effects on the nuclease resistance of oligonucleotides or stability of duplexes formed by such oligonucleotides (see, e.g., Koshkin et al., J. Am. Chem. Soc., 120:13252-3), we have found that it is not possible to predict which of any particular known modification would be most useful for any given alteration event, including for the construction of gene conversion oligonucleotides, because of the interaction of different as yet unidentified proteins during the gene alteration event. Herein, a variety of nucleic acid analogs have been developed that increase the nuclease resistance of oligonucleotides that contain them, including, e.g., nucleotides containing phosphorothioate linkages or 2'-O-methyl analogs. We recently discovered that single-stranded DNA oligonucleotides modified to contain 2'-O-methyl RNA nucleotides or phosphorothioate linkages can enable specific alteration of genetic information at a higher level than either unmodified single-stranded DNA or a chimeric RNA/DNA molecule. See priority applications incorporated herein in their entirety; see also Gamper et al., Nucleic Acids Research 28: 4332-4339 (2000). We also found that additional nucleic acid analogs which increase the nuclease resistance of oligonucleotides that contain them, including, e.g., "locked nucleic acids" or "LNAs", xylo-LNAs and L-ribo-LNAs; see, for example, Wengel & Nielsen, WO 99/14226; Wengel, WO 00/56748 and Wengel, WO 00/66604; also allow specific targeted alteration of genetic information.

The assay allows for determining the optimum length of the oligonucleotide, optimum sequence of the oligonucleotide, optimum position of the mismatched base or bases, optimum chemical

modification or modifications, optimum strand targeted for identifying and selecting the most efficient oligonucleotide for a particular gene alteration event by comparing to a control oligonucleotide. Control oligonucleotides may include a chimeric RNA-DNA double hairpin oligonucleotide directing the same gene alteration event, an oligonucleotide that matches its target completely, an oligonucleotide in which all linkages are phosphorothiolated, an oligonucleotide fully substituted with 2'-O-methyl analogs or an RNA oligonucleotide. Such control oligonucleotides either fail to direct a targeted alteration or do so at a lower efficiency as compared to the oligonucleotides of the invention. The assay further allows for determining the optimum position of a gene alteration event within an oligonucleotide, optimum concentration of the selected oligonucleotide for maximum alteration efficiency by systematically testing a range of concentrations, as well as optimization of either the source of cell extract by testing different organisms or strains, or testing cells derived from different organisms or strains, or cell lines. Using a series of single-stranded oligonucleotides, comprising all RNA or DNA residues and various mixtures of the two, several new structures are identified as viable molecules in nucleotide conversion to direct or repair a genomic mutagenic event. When extracts from mammalian, plant and fungal cells are used and are analyzed using a genetic readout assay in bacteria, single-stranded oligonucleotides having one of several modifications are found to be more active than a control RNA-DNA double hairpin chimera structure when evaluated using an in vitro gene repair assay. Similar results are also observed in vivo using yeast, mammalian, rodent, monkey, human and embryonic cells, including stem cells. Molecules containing various lengths of modified bases were found to possess greater activity than unmodified single-stranded DNA molecules.

Detailed Description Of The Invention

The present invention provides oligonucleotides having chemically modified, nuclease resistant residues, preferably at or near the termini of the oligonucleotides, and methods for their identification and use in targeted alteration of genetic material, including gene mutation, targeted gene repair and gene knockout. The oligonucleotides are preferably used for mismatch repair or alteration by changing at least one nucleic acid base, or for frameshift repair or alteration by addition or deletion of at least one nucleic acid base. The oligonucleotides of the invention direct any such alteration, including gene correction, gene repair or gene mutation and can be used, for example, to introduce a polymorphism or haplotype or to eliminate ("knockout") a particular protein activity.

The oligonucleotides of the invention are designed as substrates for homologous pairing and repair enzymes and as such have a unique backbone composition that differs from chimeric RNA-

DNA double hairpin oligonucleotides, antisense oligonucleotides, and/or other poly- or oligo-nucleotides used for altering genomic DNA, such as triplex forming oligonucleotides. The single-stranded oligonucleotides described herein are inexpensive to synthesize and easy to purify. In side-by-side comparisons, an optimized single-stranded oligonucleotide comprising modified residues as described herein is significantly more efficient than a chimeric RNA-DNA double hairpin oligonucleotide in directing a base substitution or frameshift mutation in a cell-free extract assay.

We have discovered that single-stranded oligonucleotides having a DNA domain surrounding the targeted base, with the domain preferably central to the poly- or oligo-nucleotide, and having at least one modified end, preferably at the 3' terminal region are able to alter a target genetic sequence and with an efficiency that is higher than chimeric RNA-DNA double hairpin oligonucleotides disclosed in US Patent 5,565,350. Oligonucleotides of the invention can efficiently be used to introduce targeted alterations in a genetic sequence of DNA in the presence of human, animal, plant, fungal (including yeast) proteins and in cultured cells of human liver, lung, colon, cervix, kidney, epethelium and cancer cells and in monkey, hamster, rat and mouse cells of different types, as well as embryonic stem cells. Cells for use in the invention include, e.g., fungi including *S. cerevisiae*, *Ustillago maydis* and *Candida albicans*, mammalian, mouse, hamster, rat, monkey, human and embryonic cells including stem cells. The DNA domain is preferably fully complementary to one strand of the gene target, except for the mismatch base or bases responsible for the gene alteration or conversion events. On either side of the preferably central DNA domain, the contiguous bases may be either RNA bases or, preferably, are primarily DNA bases. The central DNA domain is generally at least 8 nucleotides in length. The base(s) targeted for alteration in the most preferred embodiments are at least about 8, 9 or 10 bases from one end of the oligonucleotide.

According to certain embodiments, the termini of the oligonucleotides of the present invention comprise phosphorothioate modifications, LNA backbone modifications, or 2'-O-methyl base analogs, or any combination of these modifications. Oligonucleotides comprising 2'-O-methyl or LNA analogs are a mixed DNA/RNA polymer. These oligonucleotides are, however, single-stranded and are not designed to form a stable internal duplex structure within the oligonucleotide. The efficiency of gene alteration is surprisingly increased with oligonucleotides having internal complementary sequence comprising phosphorothioate modified bases as compared to 2'-O-methyl modifications. This result indicates that specific chemical interactions are involved between the converting oligonucleotide and the proteins involved in the conversion. The effect of other such chemical interactions to produce nuclease resistant termini using modifications other than LNA, phosphorothioate linkages, or 2'-O-methyl analog

incorporation into an oligonucleotide can not yet be predicted because the proteins involved in the alteration process and their particular chemical interaction with the oligonucleotide substituents are not yet known and cannot be predicted.

In the examples, correcting oligonucleotides of defined sequence are provided for correction of genes mutated in human diseases. In the tables of these examples, the oligonucleotides of the invention are not limited to the particular sequences disclosed. The oligonucleotides of the invention include extensions of the appropriate sequence of the longer 120 base oligonucleotides which can be added base by base to the smallest disclosed oligonucleotides of 17 bases. Thus the oligonucleotides of the invention include for each correcting change, oligonucleotides of length 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115, 116, 117, 118, 119, or 120 with further single-nucleotide additions up to the longest sequence disclosed. Moreover, the oligonucleotides of the invention do not require a symmetrical extension on either side of the central DNA domain. Similarly, the oligonucleotides of the invention as disclosed in the various tables for correction of human diseases contain phosphorothioate linkages, 2'-O-methyl analogs or LNAs or any combination of these modifications just as the assay oligonucleotides do.

The present invention, however, is not limited to oligonucleotides that contain any particular nuclease resistant modification. Oligonucleotides of the invention may be altered with any combination of additional LNAs, phosphorothioate linkages or 2'-O-methyl analogs to maximize conversion efficiency. For oligonucleotides of the invention that are longer than about 17 to about 25 bases in length, internal as well as terminal region segments of the backbone may be altered. Alternatively, simple fold-back structures at each end of a oligonucleotide or appended end groups may be used in addition to a modified backbone for conferring additional nuclease resistance.

The different oligonucleotides of the present invention preferably contain more than one of the aforementioned backbone modifications at each end. In some embodiments, the backbone modifications are adjacent to one another. However, the optimal number and placement of backbone modifications for any individual oligonucleotide will vary with the length of the oligonucleotide and the particular type of backbone modification(s) that are used. If constructs of identical sequence having phosphorothioate linkages are compared, 2, 3, 4, 5, or 6 phosphorothioate linkages at each end are preferred. If constructs of identical sequence having 2'-O-methyl base analogs are compared, 1, 2, 3 or 4

analogs are preferred. The optimal number and type of backbone modifications for any particular oligonucleotide useful for altering target DNA may be determined empirically by comparing the alteration efficiency of the oligonucleotide comprising any combination of the modifications to a control molecule of comparable sequence using any of the assays described herein. The optimal position(s) for oligonucleotide modifications for a maximally efficient altering oligonucleotide can be determined by testing the various modifications as compared to control molecule of comparable sequence in one of the assays disclosed herein. In such assays, a control molecule includes, e.g., a completely 2'-O-methyl substituted molecule, a completely complementary oligonucleotide, or a chimeric RNA-DNA double hairpin.

Increasing the number of phosphorothioate linkages, LNAs or 2'-O-methyl bases beyond the preferred number generally decreases the gene repair activity of a 25 nucleotide long oligonucleotide. Based on analysis of the concentration of oligonucleotide present in the extract after different time periods of incubation, it is believed that the terminal modifications impart nuclease resistance to the oligonucleotide thereby allowing it to survive within the cellular environment. However, this may not be the only possible mechanism by which such modifications confer greater efficiency of conversion. For example, as disclosed herein, certain modifications to oligonucleotides confer a greater improvement to the efficiency of conversion than other modifications.

Efficiency of conversion is defined herein as the percentage of recovered substate molecules that have undergone a conversion event. Depending on the nature of the target genetic material, e.g. the genome of a cell, efficiency could be represented as the proportion of cells or clones containing an extrachromosomal element that exhibit a particular phenotype. Alternatively, representative samples of the target genetic material can be sequenced to determine the percentage that have acquired the desire change. The oligonucleotides of the invention in different embodiments can alter DNA one, two, three, four, five, six, seven, eight, nine, ten, twelve, fifteen, twenty, thirty, and fifty or more fold more than control oligonucleotides. Such control oligonucleotides are oligonucleotides with fully phosphorothiolated linkages, oligonucleotides that are fully substituted with 2'-O-methyl analogs, a perfectly matched oligonucleotide that is fully complementary to a target sequence or a chimeric DNA-RNA double hairpin oligonucleotide such as disclosed in US Patent 5,565,350.

In addition, for a given oligonucleotide length, additional modifications interfere with the ability of the oligonucleotide to act in concert with the cellular recombination or repair enzyme machinery which is necessary and required to mediate a targeted substitution, addition or deletion event in DNA. For

example, fully phosphorothiolated or fully 2-O-methylated molecules are inefficient in targeted gene alteration.

The oligonucleotides of the invention as optimized for the purpose of targeted alteration of genetic material, including gene knockout or repair, are different in structure from antisense oligonucleotides that may possess a similar mixed chemical composition backbone. The oligonucleotides of the invention differ from such antisense oligonucleotides in chemical composition, structure, sequence, and in their ability to alter genomic DNA. Significantly, antisense oligonucleotides fail to direct targeted gene alteration. The oligonucleotides of the invention may target either the Watson or the Crick strand of DNA and can include any component of the genome including, for example, intron and exon sequences. The preferred embodiment of the invention is a modified oligonucleotide that binds to the non-transcribed strand of a genomic DNA duplex. In other words, the preferred oligonucleotides of the invention target the sense strand of the DNA, i.e. the oligonucleotides of the invention are complementary to the non-transcribed strand of the target duplex DNA. The sequence of the non-transcribed strand of a DNA duplex is found in the mRNA produced from that duplex, given that mRNA uses uracil-containing nucleotides in place of thymine-containing nucleotides.

Moreover, the initial observation that single-stranded oligonucleotides comprising these modifications and lacking any particular triplex forming domain have reproducibly enhanced gene repair activity in a variety of assay systems as compared to a chimeric RNA-DNA double-stranded hairpin control or single-stranded oligonucleotides comprising other backbone modifications was surprising. The single-stranded molecules of the invention totally lack the complementary RNA binding structure that stabilizes a normal chimeric double-stranded hairpin of the type disclosed in U.S. Patent 5,565,350 yet is more effective in producing targeted base conversion as compared to such a chimeric RNA-DNA double-stranded hairpin. In addition, the molecules of the invention lack any particular triplex forming domain involved in Hoogsteen interactions with the DNA double helix and required by other known oligonucleotides in other oligonucleotide dependant gene conversion systems. Although the lack of these functional domains was expected to decrease the efficiency of an alteration in a sequence, just the opposite occurs: the efficiency of sequence alteration using the modified oligonucleotides of the invention is higher than the efficiency of sequence alteration using a chimeric RNA-DNA hairpin targeting the same sequence alteration. Moreover, the efficiency of sequence alteration or gene conversion directed by an unmodified oligonucleotide is many times lower as compared to a control chimeric RNA-DNA molecule or the modified oligonucleotides of the invention targeting the same sequence alteration. Similarly,

molecules containing at least 3 2'-O-methyl base analogs are about four to five fold less efficient as compared to an oligonucleotide having the same number of phosphorothioate linkages.

The oligonucleotides of the present invention for alteration of a single base are about 17 to about 121 nucleotides in length, preferably about 17 to about 74 nucleotides in length. Most preferably, however, the oligonucleotides of the present invention are at least about 25 bases in length, unless there are self-dimerization structures within the oligonucleotide. If the oligonucleotide has such an unfavorable structure, lengths longer than 35 bases are preferred. Oligonucleotides with modified ends both shorter and longer than certain of the exemplified, modified oligonucleotides herein function as gene repair or gene knockout agents and are within the scope of the present invention.

Once an oligomer is chosen, it can be tested for its tendency to self-dimerize, since self-dimerization may result in reduced efficiency of alteration of genetic information. Checking for self-dimerization tendency can be accomplished manually or, more preferably, by using a software program. One such program is Oligo Analyzer 2.0, available through Integrated DNA Technologies (Coralville, IA 52241) (http://www.idtdna.com); this program is available for use on the world wide web at

http://www.idtdna.com/program/oligoanalyzer/

oligoanalyzer.asp.

For each oligonucleotide sequence input into the program, Oligo Analyzer 2.0 reports possible self-dimerized duplex forms, which are usually only partially duplexed, along with the free energy change associated with such self-dimerization. Delta G-values that are negative and large in magnitude, indicating strong self-dimerization potential, are automatically flagged by the software as "bad". Another software program that analyzes oligomers for pair dimer formation is Primer Select from DNASTAR, Inc., 1228 S. Park St., Madison, WI 53715, Phone: (608) 258-7420 (http://www.dnastar.com/products/PrimerSelect.html).

If the sequence is subject to significant self-dimerization, the addition of further sequence flanking the "repair" nucleotide can improve gene correction frequency.

Generally, the oligonucleotides of the present invention are identical in sequence to one strand of the target DNA, which can be either strand of the target DNA, with the exception of one or more targeted bases positioned within the DNA domain of the oligonucleotide, and preferably toward the middle between the modified terminal regions. Preferably, the difference in sequence of the oligonucleotide as compared to the targeted genomic DNA is located at about the middle of the oligonucleotide sequence. In a preferred embodiment, the oligonucleotides of the invention are complementary to the non-transcribed strand of a duplex. In other words, the preferred oligonucleotides target the sense strand of the DNA, i.e.

the oligonucleotides of the invention are preferably complementary to the strand of the target DNA the sequence of which is found in the mRNA.

The oligonucleotides of the invention can include more than a single base change. In an oligonucleotide that is about a 70-mer, with at least one modified residue incorporated on the ends, as disclosed herein, multiple bases can be simultaneously targeted for change. The target bases may be up to 27 nucleotides apart and may not be changed together in all resultant plasmids in all cases. There is a frequency distribution such that the closer the target bases are to each other in the central DNA domain within the oligonucleotides of the invention, the higher the frequency of change in a given cell. Target bases only two nucleotides apart are changed together in every case that has been analyzed. The farther apart the two target bases are, the less frequent the simultaneous change. Thus, oligonucleotides of the invention may be used to repair or alter multiple bases rather than just one single base. For example, in a 74-mer oligonucleotide having a central base targeted for change, a base change event up to about 27 nucleotides away can also be effected. The positions of the altering bases within the oligonucleotide can be optimized using any one of the assays described herein. Preferably, the altering bases are at least about 8 nucleotides from one end of the oligonucleotide.

The oligonucleotides of the present invention can be introduced into cells by any suitable means. According to certain preferred embodiments, the modified oligonucleotides may be used alone. Suitable means, however, include the use of polycations, cationic lipids, liposomes, polyethylenimine (PEI), electroporation, biolistics, microinjection and other methods known in the art to facilitate cellular uptake. According to certain preferred embodiments of the present invention, the isolated cells are treated in culture according to the methods of the invention, to mutate or repair a target gene. Modified cells may then be reintroduced into the organism as, for example, in bone marrow having a targeted gene. Alternatively, modified cells may be used to regenerate the whole organism as, for example, in a plant having a desired targeted genomic change. In other instances, targeted genomic alteration, including repair or mutagenesis, may take place in vivo following direct administration of the modified, single-stranded oligonucleotides of the invention to a subject.

The single-stranded, modified oligonucleotides of the present invention have numerous applications as gene repair, gene modification, or gene knockout agents. Such oligonucleotides may be advantageously used, for example, to introduce or correct multiple point mutations. Each mutation leads to the addition, deletion or substitution of at least one base pair. The methods of the present invention offer distinct advantages over other methods of altering the genetic makeup of an organism, in that only the individually targeted bases are altered. No additional foreign DNA sequences are added to the

genetic complement of the organism. Such agents may, for example, be used to develop plants or animals with improved traits by rationally changing the sequence of selected genes in cultured cells. Modified cells are then cloned into whole plants or animals having the altered gene. See, e.g., U.S. Patent 6,046,380 and U.S. Patent 5,905,185 incorporated hererin by reference. Such plants or animals produced using the compositions of the invention lack additional undesirable selectable markers or other foreign DNA sequences. Targeted base pair substitution or frameshift mutations introduced by an oligonucleotide in the presence of a cell-free extract also provides a way to modify the sequence of extrachromosomal elements, including, for example, plasmids, cosmids and artificial chromosomes. The oligonucleotides of the invention also simplify the production of transgenic animals having particular modified or inactivated genes. Altered animal or plant model systems such as those produced using the methods and oligonucleotides of the invention are invaluable in determining the function of a gene and in evaluating drugs. The oligonucleotides and methods of the present invention may also be used for gene therapy to correct mutations causative of human diseases.

The purified oligonucleotide compositions may be formulated in accordance with routine procedures as a pharmaceutical composition adapted for bathing cells in culture, for microinjection into cells in culture, and for intravenous administration to human beings or animals. Typically, compositions for cellular administration or for intravenous administration into animals, including humans, are solutions in sterile isotonic aqueous buffer. Where necessary, the composition may also include a solubilizing agent and a local anaesthetic such as lignocaine to ease pain at the site of the injection. Generally, the ingredients will be supplied either separately or mixed together in unit dosage form, for example, as a dry, lyophilized powder or water-free concentrate. The composition may be stored in a hermetically sealed container such as an ampule or sachette indicating the quantity of active agent in activity units. Where the composition is administered by infusion, it can be dispensed with an infusion bottle containing sterile pharmaceutical grade "water for injection" or saline. Where the composition is to be administered by injection, an ampule of sterile water for injection or saline may be provided so that the ingredients may be mixed prior to administration.

Pharmaceutical compositions of this invention comprise the compounds of the present invention and pharmaceutically acceptable salts thereof, with any pharmaceutically acceptable ingredient, excipient, carrier, adjuvant or vehicle.

The oligonucleotides of the invention are preferably administered to the subject in the form of an injectable composition. The composition is preferably administered parenterally, meaning intravenously, intraarterially, intrathecally, interstitially or intracavitarilly. Pharmaceutical compositions of

this invention can be administered to mammals including humans in a manner similar to other diagnostic or therapeutic agents. The dosage to be administered, and the mode of administration will depend on a variety of factors including age, weight, sex, condition of the subject and genetic factors, and will ultimately be decided by medical personnel subsequent to experimental determinations of varying dosage as described herein. In general, dosage required for correction and therapeutic efficacy will range from about 0.001 to 50,000 µg/kg, preferably between 1 to 250 µg/kg of host cell or body mass, and most preferably at a concentration of between 30 and 60 micromolar.

For cell administration, direct injection into the nucleus, biolistic bombardment, electroporation, liposome transfer and calcium phosphate precipitation may be used. In yeast, lithium acetate or spheroplast transformation may also be used. In a preferred method, the administration is performed with a liposomal transfer compound, e.g., DOTAP (Boehringer-Mannheim) or an equivalent such as lipofectin. The amount of the oligonucleotide used is about 500 nanograms in 3 micrograms of DOTAP per 100,000 cells. For electroporation, between 20 and 2000 nanograms of oligonucleotide per million cells to be electroporated is an appropriate range of dosages which can be increased to improve efficiency of genetic alteration upon review of the appropriate sequence according to the methods described herein.

Another aspect of the invention is a kit comprising at least one oligonucleotide of the invention. The kit may comprise an addition reagent or article of manufacture. The additional reagent or article of manufacture may comprise a cell extract, a cell, or a plasmid, such as one of those disclosed in the Figures herein, for use in an assay of the invention.

Brief Description Of The Drawings

Figure 1. *Flow diagram for the generation of modified single-stranded oligonucleotides.* The upper strands of chimeric oligonucleotides I and II are separated into pathways resulting in the generation of single-stranded oligonucleotides that contain (A) 2'-O-methyl RNA nucleotides or (B) phosphorothioate linkages. Fold changes in repair activity for correction of kan^s in the HUH7 cell-free extract are presented in parenthesis. HUH7 cells are described in Nakabayashi et al., Cancer Research 42: 3858-3863 (1982). Each single-stranded oligonucleotide is 25 bases in length and contains a G residue mismatched to the complementary sequence of the kan^s gene. The numbers 3, 6, 8, 10, 12 and 12.5 respectively indicate how many phosphorothioate linkages (S) or 2'-O-methyl RNA nucleotides (R) are at each end of the molecule. Hence oligo 12S/25G contains an all phosphorothioate backbone, displayed as a dotted line. Smooth lines indicate DNA residues, wavy lines indicate 2'-O-methyl RNA

residues and the carat indicates the mismatched base site (G). Figure 1(C) provides a schematic plasmid indicating the sequence of the kan chimeric double-stranded hairpin oligonucleotide (left) and the sequence the tet chimeric double-stranded hairpin oligonucleotide used in other experiments. Figure 1(D) provides a flow chart of a kan experiment in which a chimeric double-stranded hairpin oligonucleotide is used.

Figure 2. *Genetic readout system for correction of a point mutation in plasmid* pK^{s}m4021. A mutant kanamycin gene harbored in plasmid pK^{s}m4021 is the target for correction by oligonucleotides. The mutant G is converted to a C by the action of the oligo. Corrected plasmids confer resistance to kanamycin in *E.coli* (DH10B) after electroporation leading to the genetic readout and colony counts.

Figure 3: *Target plasmid and sequence correction of a frameshift mutation by chimeric and single-stranded oligonucleotides.* (A) Plasmid pT$^s\Delta$208 contains a single base deletion mutation at position 208 rendering it unable to confer tet resistance. The target sequence presented below indicates the insertion of a T directed by the oligonucleotides to re-establish the resistant phenotype. (B) DNA sequence confirming base insertion directed by Tet 3S/25G; the yellow highlight indicates the position of frameshift repair.

Figure 4. *DNA sequences of representative kan^r colonies.* Confirmation of sequence alteration directed by the indicated molecule is presented along with a table outlining codon distribution. Note that 10S/25G and 12S/25G elicit both mixed and unfaithful gene repair. The number of clones sequenced is listed in parentheses next to the designation for the single-stranded oligonucleotide. A plus (+) symbol indicates the codon identified while a figure after the (+) symbol indicates the number of colonies with a particular sequence. TAC/TAG indicates a mixed peak. Representative DNA sequences are presented below the table with yellow highlighting altered residues.

Figure 5. *Gene correction in HeLa cells.* Representative oligonucleotides of the invention are co-transfected with the pCMVneo(-)FlAsH plasmid (shown in Figure 9) into HeLa cells. Ligand is diffused into cells after co-transfection of plasmid and oligonucleotides. Green fluorescence indicates gene correction of the mutation in the antibiotic resistance gene. Correction of the mutation results in the expression of a fusion protein that carries a marker ligand binding site and when the fusion protein binds the ligand, a green fluorescence is emitted. The ligand is produced by Aurora Biosciences and can readily diffuse into cells enabling a measurement of corrected protein function; the protein must bind the ligand directly to induce fluorescence. Hence cells bearing the corrected plasmid gene appear green while "uncorrected" cells remain colorless.

Figure 6. *Z-series imaging of corrected cells.* Serial cross-sections of the HeLa cell represented in Figure 5 are produced by Zeiss 510 LSM confocal microscope revealing that the fusion protein is contained within the cell.

Figure 7. *Hygromycin-eGFP target plasmids.* (A) Plasmid pAURHYG(ins)GFP contains a single base insertion mutation between nucleotides 136 and 137, at codon 46, of the Hygromycin B coding sequence (cds) which is transcribed from the constitutive ADH1 promoter. The target sequence presented below indicates the deletion of an A and the substitution of a C for a T directed by the oligonucleotides to re-establish the resistant phenotype. (B) Plasmid pAURHYG(rep)GFP contains a base substitution mutation introducing a G at nucleotide 137, at codon 46, of the Hygromycin B coding sequence (cds). The target sequence presented below the diagram indicates the amino acid conservative replacement of G with C, restoring gene function.

Figure 8. *Oligonucleotides for correction of hygromycin resistance gene.* The sequence of the oligonucleotides used in experiments to assay correction of a hygromycin resistance gene are shown. DNA residues are shown in capital letters, RNA residues are shown in lowercase and nucleotides with a phosphorothioate backbone are capitalized and underlined.

Figure 9. *pAURNeo(-)FlAsH plasmid.* This figure describes the plasmid structure, target sequence, oligonucleotides, and the basis for detection of the gene alteration event by fluorescence.

Figure 10. pYESHyg(x)eGFP plasmid. This plasmid is a construct similar to the pAURHyg(x)eGFP construct shown in Figure 7, except the promoter is the inducible GAL1 promoter. This promoter is inducible with galactose, leaky in the presence of raffinose, and repressed in the presence of dextrose.

The following examples are provided by way of illustration only, and are not intended to limit the scope of the invention disclosed herein.

EXAMPLE 1
Assay Method For Base Alteration And Preferred Oligonucleotide Selection

In this example, single-stranded and double-hairpin oligonucleotides with chimeric backbones (see Figure 1 for structures (A and B) and sequences (C and D) of assay oligonucleotides) are used to correct a point mutation in the kanamycin gene of $pK^{s}m4021$ (Figure 2) or the tetracycline gene of $pT^{s}\Delta 208$ (Figure 3). All kan oligonucleotides share the same 25 base sequence surrounding the target base identified for change, just as all tet oligonucleotides do. The sequence is given in Figures 1C and Figure 1D. Each plasmid contains a functional ampicillin gene. Kanamycin gene function is restored

when a G at position 4021 is converted to a C (via a substitution mutation); tetracycline gene function is restored when a deletion at position 208 is replaced by a C (via frameshift mutation). A separate plasmid, pAURNeo(-)FlAsH (Figure 9), bearing the kan^S gene is used in the cell culture experiments. This plasmid was constructed by inserting a synthetic expression cassette containing a neomycin phosphotransferase (kanamycin resistance) gene and an extended reading frame that encodes a receptor for the FlAsH ligand into the pAUR123 shuttle vector (Panvera Corp., Madison, WI). The resulting construct replicates in *S. cerevisiae* at low copy number, confers resistance to aureobasidinA and constitutively expresses either the Neo+/FlAsH fusion product (after alteration) or the truncated Neo-/FlAsH product (before alteration) from the ADH1 promoter. By extending the reading frame of this gene to code for a unique peptide sequence capable of binding a small ligand to form a fluorescent complex, restoration of expression by correction of the stop codon can be detected in real time using confocal microscopy. Additional constructs can be made to test additional gene alteration events.

We also construct three mammalian expression vectors, pHyg(rep)eGFP, pHyg(Δ)eGFP, pHyg(ins)eGFP, that contain a substitution mutation at nucleotide 137 of the hygromycin-B coding sequence. (rep) indicates a T137⇨G replacement, (Δ) represents a deletion of the G137 and (ins) represents an A insertion between nucleotides 136 and 137. All point mutations create a nonsense termination codon at residue 46. We use pHygEGFP plasmid (Invitrogen, CA) DNA as a template to introduce the mutations into the hygromycin-eGFP fusion gene by a two step site-directed mutagenesis PCR protocol. First, we generate overlapping 5' and a 3' amplicons surrounding the mutation site by PCR for each of the point mutation sites. A 215 bp 5' amplicon for the (rep), (Δ) or (ins) was generated by polymerization from oligonucleotide primer HygEGFPf (5'-AATACGACTCACTATAGG-3') to primer Hygrepr (5'GACCTATCCACGCCCTCC-3'), HygΔr (5'-GACTATCCACGCCCTCC-3'), or Hyginsr (5'-GACATTATCCACGCCCTCC-3'), respectively. We generate a 300bp 3' amplicon for the (rep), (Δ) or (ins) by polymerization from oligonucleotide primers Hygrepf (5'-CTGGGATAGGTCCTGCGG-3'), HygΔf (5'-CGTGGATAGTCCTGCGG-3'), Hyginsf (5'-CGTGGATAATGTCCTGCGG-3'), respectively to primer HygEGFPr (5'-AAATCACGCCATGTAGTG-3'). We mix 20 ng of each of the resultant 5' and 3' overlapping amplicon mutation sets and use the mixture as a template to amplify a 523 bp fragment of the Hygromycin gene spanning the KpnI and RsrII restriction endonuclease sites. We use the Expand PCR system (Roche) to generate all amplicons with 25 cycles of denaturing at 94°C for 10 seconds, annealing at 55°C for 20 seconds and elongation at 68°C for 1 minute. We digest 10 µg of vector pHygEGFP and 5 µg of the resulting fragments for each mutation with KpnI and RsrII (NEB) and gel purify the fragment for enzymatic ligation. We ligate each mutated insert into pHygEGFP vector at 3:1 molar ration using T4

DNA ligase (Roche). We screen clones by restriction digest, confirm the mutation by Sanger dideoxy chain termination sequencing and purify the plasmid using a Qiagen maxiprep kit.

Oligonucleotide synthesis and cells. Chimeric oligonucleotides and single-stranded oligonucleotides (including those with the indicated modifications) are synthesized using available phosphoramidites on controlled pore glass supports. After deprotection and detachment from the solid support, each oligonucleotide is gel-purified using, for example, procedures such as those described in Gamper *et al.*, *Biochem.* 39, 5808-5816 (2000) and the concentrations determined spectrophotometrically (33 or 40 µg/ml per A_{260} unit of single-stranded or hairpin oligomer). HUH7 cells are grown in DMEM, 10% FBS, 2mM glutamine, 0.5% pen/strep. The *E.coli* strain, DH10B, is obtained from Life Technologies (Gaithersburg, MD); DH10B cells contain a mutation in the RECA gene (*rec*A).

Cell-free extracts. We prepare cell-free extracts from HUH7 cells or other mammalian cells, as follows. We employ this protocol with essentially any mammalian cell including, for example, H1299 cells (human epithelial carcinoma, non-small cell lung cancer), C127I (immortal murine mammary epithelial cells), MEF (mouse embryonic fibroblasts), HEC-1-A (human uterine carcinoma), HCT15 (human colon cancer), HCT116 (human colon carcinoma), LoVo (human colon adenocarcinoma), and HeLa (human cervical carcinoma). We harvest approximately $2x10^8$ cells. We then wash the cells immediately in cold hypotonic buffer (20 mM HEPES, pH7.5; 5 mM KCl; 1.5 mM $MgCl_2$; 1 mM DTT) with 250 mM sucrose. We then resuspend the cells in cold hypotonic buffer without sucrose and after 15 minutes we lyse the cells with 25 strokes of a Dounce homogenizer using a tight fitting pestle. We incubate the lysed cells for 60 minutes on ice and centrifuge the sample for 15 minutes at 12000x*g*. The cytoplasmic fraction is enriched with nuclear proteins due to the extended co-incubation of the fractions following cell breakage. We then immediately aliquote and freeze the supernatant at -80°C. We determine the protein concentration in the extract by the Bradford assay.

We also perform these experiments with cell-free extracts obtained from fungal cells, including, for example, *S. cerevisiae* (yeast), *Ustilago maydis*, and *Candida albicans*. For example, we grow yeast cells into log phase in 2L YPD medium for 3 days at 30°C. We then centrifuge the cultures at 5000x*g*, resuspend the pellets in a 10% sucrose, 50 mM Tris, 1mM EDTA lysis solution and freeze them on dry ice. After thawing, we add KCl, spermidine and lyticase to final concentrations of 0.25 mM, 5 mM and 0.1 mg/ml, respectively. We incubate the suspension on ice for 60 minutes, add PMSF and Triton X100 to final concentrations of 0.1 mM and 0.1% and continue to incubate on ice for 20 minutes. We centrifuge the lysate at 3000x*g* for 10 minutes to remove larger debris. We then remove the supernatant and clarify it by centrifuging at 30000x*g* for 15 minutes. We then add glycerol to the clarified extract to a

concentration of 10% (v/v) and freeze aliquots at -80°C. We determine the protein concentration of the extract by the Bradford assay.

Reaction mixtures of 50 µl are used, consisting of 10-30 µg protein of cell-free extract, which can be optionally substituted with purified proteins or enriched fractions, about 1.5 µg chimeric double-hairpin oligonucleotide or 0.55 µg single-stranded molecule (3S/25G or 6S/25G, see Figure 1), and 1 µg of plasmid DNA (see Figures 2 and 3) in a reaction buffer of 20 mM Tris, pH 7.4, 15 mM $MgCl_2$, 0.4 mM DTT, and 1.0 mM ATP. Reactions are initiated with extract and incubated at 30°C for 45 min. The reaction is stopped by placing the tubes on ice and then immediately deproteinized by two phenol/chloroform (1:1) extractions. Samples are then ethanol precipitated. The nucleic acid is pelleted at 15,000 r.p.m. at 4°C for 30 min., is washed with 70% ethanol, resuspended in 50 µl H_20, and is stored at -20°C. 5 µl of plasmid from the resuspension (~100 ng) was transfected in 20 µl of DH10B cells by electroporation (400 V, 300 µF, 4 kΩ) in a Cell-Porator apparatus (Life Technologies). After electroporation, cells are transferred to a 14 ml Falcon snap-cap tube with 2 ml SOC and shaken at 37°C for 1 h. Enhancement of final kan colony counts is achieved by then adding 3 ml SOC with 10 µg/ml kanamycin and the cell suspension is shaken for a further 2 h at 37°C. Cells are then spun down at 3750 x g and the pellet is resuspended in 500 µl SOC. 200 µl is added undiluted to each of two kanamycin (50 µg/ml) agar plates and 200 µl of a 10^5 dilution is added to an ampicillin (100 µg/ml) plate. After overnight 37°C incubation, bacterial colonies are counted using an Accucount 1000 (Biologics). Gene conversion effectiveness is measured as the ratio of the average of the kan colonies on both plates per amp colonies multiplied by 10^{-5} to correct for the amp dilution.

The following procedure can also be used. 5 µl of resuspended reaction mixtures (total volume 50 µl) are used to transform 20 µl aliquots of electro-competent ΔH10B bacteria using a Cell-Porator apparatus (Life Technologies). The mixtures are allowed to recover in 1 ml SOC at 37°C for 1 hour at which time 50 µg/ml kanamycin or 12 µg/ml tetracycline is added for an additional 3 hours. Prior to plating, the bacteria are pelleted and resuspended in 200 µ1 of SOC. 100 µl aliquots are plated onto kan or tet agar plates and 100 µl of a 10^{-4} dilution of the cultures are concurrently plated on agar plates containing 100 µg/ml of ampicillin. Plating is performed in triplicate using sterile Pyrex beads. Colony counts are determined by an Accu-count 1000 plate reader (Biologics). Each plate contains 200-500 ampicillin resistant colonies or 0-500 tetracycline or kanamycin resistant colonies. Resistant colonies are selected for plasmid extraction and DNA sequencing using an ABI Prism kit on an ABI 310 capillary sequencer (PE Biosystems).

Chimeric single-stranded oligonucleotides. In Figure 1 the upper strands of chimeric oligonucleotides I and II are separated into pathways resulting in the generation of single-stranded oligonucleotides that contain (Figure 1A) 2'-O-methyl RNA nucleotides or (Figure 1B) phosphorothioate linkages. Fold changes in repair activity for correction of kan^s in the HUH7 cell-free extract are presented in parenthesis. Each single-stranded oligonucleotide is 25 bases in length and contains a G residue mismatched to the complementary sequence of the kan^s gene.

Molecules bearing 3, 6, 8, 10 and 12 phosphorothioate linkages in the terminal regions at each end of a backbone with a total of 24 linkages (25 bases) are tested in the kan^s system. Alternatively, molecules bearing 2, 4, 5, 7, 9 and 11 in the terminal regions at each end are tested. The results of one such experiment, presented in Table 1 and Figure 1B, illustrate an enhancement of correction activity directed by some of these modified structures. In this illustrative example, the most efficient molecules contained 3 or 6 phosphorothioate linkages at each end of the 25-mer; the activities are approximately equal (molecules IX and X with results of 3.09 and 3.7 respectively). A reduction in alteration activity may be observed as the number of modified linkages in the molecule is further increased. Interestingly, a single-strand molecule containing 24 phosphorothioate linkages is minimally active suggesting that this backbone modification when used throughout the molecule supports only a low level of targeted gene repair or alteration. Such a non-altering, completely modified molecule can provide a baseline control for determining efficiency of correction for a specific oligonucleotide molecule of known sequence in defining the optimum oligonucleotide for a particular alteration event.

The efficiency of gene repair directed by phosphorothioate-modified, single-stranded molecules, in a length dependent fashion, led us to examine the length of the RNA modification used in the original chimera as it relates to correction. Construct III represents the "RNA-containing" strand of chimera I and, as shown in Table 1 and Figure 2A, it promotes inefficient gene repair. But, as shown in the same figure, reducing the RNA residues on each end from 10 to 3 increases the frequency of repair. At equal levels of modification, however, 25-mers with 2'-O-methyl ribonucleotides were less effective gene repair agents than the same oligomers with phosphorothioate linkages. These results reinforce the fact that an RNA containing oligonucleotide is not as effective in promoting gene repair or alteration as a modified DNA oligonucleotide.

Repair of the kanamycin mutation requires a G→C exchange. To confirm that the specific desired correction alteration was obtained, colonies selected at random from multiple experiments are processed and the isolated plasmid DNA is sequenced. As seen in Figure 4, colonies generated through the action of the single-stranded molecules 3S/25G (IX), 6S/25G (X) and 8S/25G (XI) respectively

contained plasmid molecules harboring the targeted base correction. While a few colonies appeared on plates derived from reaction mixtures containing 25-mers with 10 or 12 thioate linkages on both ends, the sequences of the plasmid molecules from these colonies contain nonspecific base changes. In these illustrative examples, the second base of the codon is changed (see Figure 3). These results show that modified single-strands can direct gene repair, but that efficiency and specificity are reduced when the 25-mers contain 10 or more phosphorothioate linkages at each end.

In Figure 1, the numbers 3, 6, 8, 10, 12 and 12.5 respectively indicate how many phosphorothioate linkages (S) or 2'-O-methyl RNA nucleotides (R) are at each end of the examplified molecule although other molecules with 2, 4, 5, 7, 9 and 11 modifications at each end can also be tested. Hence oligo 12S/25G represents a 25-mer oligonucleotide which contains 12 phosphorothioate linkages on each side of the central G target mismatch base producing a fully phosphorothioate linked backbone, displayed as a dotted line. The dots are merely representative of a linkage in the figure and do not depict the actual number of linkages of the oligonucleotide. Smooth lines indicate DNA residues, wavy lines indicate 2'-O-methyl RNA residues and the carat indicates the mismatched base site (G).

Correction of a mutant kanamycin gene in cultured mammalian cells. The experiments are performed using different mammalian cells, including, for example, 293 cells (transformed human primary kidney cells), HeLa cells (human cervical carcinoma), and H1299 (human epithelial carcinoma, non-small cell lung cancer). HeLa cells are grown at 37°C and 5% CO_2 in a humidified incubator to a density of 2 x 10^5 cells/ml in an 8 chamber slide (Lab-Tek). After replacing the regular DMEM with Optimem, the cells are co-transfected with 10 µg of plasmid pAURNeo(-)FlAsH and 5 µg of modified single-stranded oligonucleotide (3S/25G) that is previously complexed with 10 µg lipofectamine, according to the manufacturer's directions (Life Technologies). The cells are treated with the liposome-DNA-oligo mix for 6 hrs at 37°C. Treated cells are washed with PBS and fresh DMEM is added. After a 16-18 hr recovery period, the culture is assayed for gene repair. The same oligonucleotide used in the cell-free extract experiments is used to target transfected plasmid bearing the kan^S gene. Correction of the point mutation in this gene eliminates a stop codon and restores full expression. This expression can be detected by adding a small non-fluorescent ligand that bound to a C-C-R-E-C-C sequence in the genetically modified carboxy terminus of the kan protein, to produce a highly fluorescent complex (FlAsH system, Aurora Biosciences Corporation). Following a 60 min incubation at room temperature with the ligand (FlAsH-EDT2), cells expressing full length kan product acquire an intense green fluorescence detectable by fluorescence microscopy using a fluorescein filter set. Similar experiments are performed using the HygeGFP target as described in Example 2 with a variety of mammalian cells, including, for

example, COS-1 and COS-7 cells (African green monkey), and CHO-K1 cells (Chinese hamster ovary). The experiments are also performed with PG12 cells (rat pheochromocytoma) and ES cells (human embryonic stem cells).

Summary of experimental results. Tables 1, 2 and 3 respectively provide data on the efficiency of gene repair directed by single-stranded oligonucleotides. Table 1 presents data using a cell-free extract from human liver cells (HUH7) to catalyze repair of the point mutation in plasmid pkan^{s}m4021 (see Figure 1). Table 2 illustrates that the oligomers are not dependent on MSH2 or MSH3 for optimal gene repair activity. Table 3 illustrates data from the repair of a frameshift mutation (Figure 3) in the tet gene contained in plasmid pTetΔ208. Table 4 illustrates data from repair of the pkan^{s}m4021 point mutation catalyzed by plant cell extracts prepared from canola and musa (banana). Colony numbers are presented as kan^{r} or tet^{r} and fold increases (single strand versus double hairpin) are presented for kan^{r} in Table 1.

Figure 5A is a confocal picture of HeLa cells expressing the corrected fusion protein from an episomal target. Gene repair is accomplished by the action of a modified single-stranded oligonucleotide containing 3 phosphorothioate linkages at each end (3S/25G). Figure 5B represents a "Z-series" of HeLa cells bearing the corrected fusion gene. This series sections the cells from bottom to top and illustrates that the fluorescent signal is "inside the cells".

Results. In summary, we have designed a novel class of single-stranded oligonucleotides with backbone modifications at the termini and demonstrate gene repair/conversion activity in mammalian and plant cell-free extracts. We confirm that the all DNA strand of the RNA-DNA double-stranded double hairpin chimera is the active component in the process of gene repair. In some cases, the relative frequency of repair by the novel oligonucleotides of the invention is elevated approximately 3-4-fold when compared to frequencies directed by chimeric RNA-DNA double hairpin oligonucleotides.

This strategy centers around the use of extracts from various sources to correct a mutation in a plasmid using a modified single-stranded or a chimeric RNA-DNA double hairpin oligonucleotide. A mutation is placed inside the coding region of a gene conferring antibiotic resistance in bacteria, here kanamycin or tetracycline. The appearance of resistance is measured by genetic readout in *E.coli* grown in the presence of the specified antibiotic. The importance of this system is that both phenotypic alteration and genetic inheritance can be measured. Plasmid pK^{s}m4021 contains a mutation (T→G) at residue 4021 rendering it unable to confer antibiotic resistance in *E.coli.* This point mutation is targeted for repair by oligonucleotides designed to restore kanamycin resistance. To avoid concerns of

plasmid contamination skewing the colony counts, the directed correction is from G→C rather than G→T (wild-type). After isolation, the plasmid is electroporated into the DH10B strain of *E.coli*, which contains inactive RecA protein. The number of kanamycin colonies is counted and normalized by ascertaining the number of ampicillin colonies, a process that controls for the influence of electroporation. The number of colonies generated from three to five independent reactions was averaged and is presented for each experiment. A fold increase number is recorded to aid in comparison.

The original RNA-DNA double hairpin chimera design, e.g., as disclosed in U.S. Patent 5,565,350, consists of two hybridized regions of a single-stranded oligonucleotide folded into a double hairpin configuration. The double-stranded targeting region is made up of a 5 base pair DNA/DNA segment bracketed by 10 base pair RNA/DNA segments. The central base pair is mismatched to the corresponding base pair in the target gene. When a molecule of this design is used to correct the kan^s mutation, gene repair is observed (I in Figure 1A). Chimera II (Figure 1B) differs partly from chimera I in that only the DNA strand of the double hairpin is mismatched to the target sequence. When this chimera was used to correct the kan^s mutation, it was twice as active. In the same study, repair function could be further increased by making the targeting region of the chimera a continuous RNA/DNA hybrid.

Frame shift mutations are repaired. By using plasmid $pT^s\Delta208$, described in Figure 1(C) and Figure 3, the capacity of the modified single-stranded molecules that showed activity in correcting a point mutation, can be tested for repair of a frameshift. To determine efficiency of correction of the mutation, a chimeric oligonucleotide (Tet I), which is designed to insert a T residue at position 208, is used. A modified single-stranded oligonucleotide (Tet IX) directs the insertion of a T residue at this same site. Figure 3 illustrates the plasmid and target bases designated for change in the experiments. When all reaction components are present (extract, plasmid, oligomer), tetracycline resistant colonies appear. The colony count increases with the amount of oligonucleotide used up to a point beyond which the count falls off (Table 3). No colonies above background are observed in the absence of either extract or oligonucleotide, nor when a modified single-stranded molecule bearing perfect complementarity is used. Figure 3 represents the sequence surrounding the target site and shows that a T residue is inserted at the correct site. We have isolated plasmids from fifteen colonies obtained in three independent experiments and each analyzed sequence revealed the same precise nucleotide insertion. These data suggest that the single-stranded molecules used initially for point mutation correction can also repair nucleotide deletions.

Comparison of phosphorothioate oligonucleotides to 2'-O-methyl substituted oligonucleotides. From a comparison of molecules VII and XI, it is apparent that gene repair is more

subject to inhibition by RNA residues than by phosphorothioate linkages. Thus, even though both of these oligonucleotides contain an equal number of modifications to impart nuclease resistance, XI (with 16 phosphorothioate linkages) has good gene repair activity while VII (with 16 2'-O-methyl RNA residues) is inactive. Hence, the original chimeric double hairpin oligonucleotide enabled correction directed, in large part, by the strand containing a large region of contiguous DNA residues.

Oligonucleotides can target multiple nucleotide alterations within the same template. The ability of individual single-stranded oligonucleotides to correct multiple mutations in a single target template is tested using the plasmid pK^{s}m4021 and the following single-stranded oligonucleotides modified with 3 phosphorothioate linkages at each end (indicated as underlined nucleotides): Oligo1 is a 25-mer with the sequence TTCGATAAGCCTATGCTGACCCGTG corrects the original mutation present in the kanamycin resistance gene of pK^{s}m4021 as well as directing another alteration 2 basepairs away in the target sequence (both indicated in boldface); Oligo2 is a 70-mer with the 5'-end sequence TTCGGCTACGACTGGGCACAACAGACAATTGGC with the remaining nucleotides being completely complementary to the kanamycin resistance gene and also ending in 3 phosphorothioate linkages at the 3' end. Oligo2 directs correction of the mutation in pK^{s}m4021 as well as directing another alteration 21 basepairs away in the target sequence (both indicated in boldface).

We also use additional oligonucleotides to assay the ability of individual oligonucleotides to correct multiple mutations in the pK^{s}M4021 plasmid. These include, for example, a second 25-mer that alters two nucleotides that are three nucleotides apart with the sequence 5'-TTGTGCCCAGTCGTATCCGAATAGC-3', a 70-mer that alters two nucleotides that are 21 nucleotides apart with the sequence 5'-CATCAGAGCAGCCAATTGTCTGTTGTGCCCAGTCGTAGCCGAA TAGCCTCTCCACCCAAGCGGCCGGAGA-3'; and another 70-mer that alters two nucleotides that are 21 nucleotides apart with the sequence 5'-GCTGACAGCCGGAACACGGCGGCATCAGAGCAGCCAATTGTCTGTTGTGCCCAGTCGTAGCCGAAT AGCCT-3'. The nucleotides in the oligonucleotides that direct alteration of the target sequence are underlined and in boldface. These oligonucleotides are modified in the same way as the other oligonucleotides of the invention.

We assay correction of the original mutation in pK^{s}m4021 by monitoring kanamycin resistance (the second alterations which are directed by Oligo2 and Oligo3 are silent with respect to the kanamycin resistance phenotype). In addition, in experiments with Oligo2, we also monitor cleavage of the resulting plasmids using the restriction enzyme Tsp509I which cuts at a specific site present only when the second alteration has occurred (at ATT in Oligo2). We then sequence these clones to

determine whether the additional, silent alteration has also been introduced. The results of an analysis are presented below:

	Oligo1 (25-mer)	Oligo2 (70-mer)
Clones with both sites changed	9	7
Clones with a single site changed	0	2
Clones that were not changed	4	1

Nuclease sensitivity of unmodified DNA oligonucleotide. Electrophoretic analysis of nucleic acid recovered from the cell-free extract reactions conducted here confirm that the unmodified single-stranded 25-mer did not survive incubation whereas greater than 90% of the terminally modified oligos did survive (as judged by photo-image analyses of agarose gels).

Plant extracts direct repair. The modified single-stranded constructs can be tested in plant cell extracts. We have observed gene alteration using extracts from multiple plant sources, including, for example, Arabidopsis, tobacco, banana, maize, soybean, canola, wheat, spinach as well as spinach chloroplast extract. We prepare the extracts by grinding plant tissue or cultured cells under liquid nitrogen with a mortar and pestle. We extract 3 ml of the ground plant tissue with 1.5 ml of extraction buffer (20 mM HEPES, pH7.5; 5 mM Kcl; 1.5 mM $MgCl_2$; 10 mM DTT; 10% [v/v] glycerol; and 1 % [w/v] PVP). We then homogenize the samples with 15 strokes of a Dounce homogenizer. Following homogenization, we incubate the samples on ice for 1 hour and centrifuge at 3000x*g* for 5 minutes to remove plant cell debris. We then determine the protein concentration in the supernatants (extracts) by Bradford assay. We dispense 100 µg (protein) aliquots of the extracts which we freeze in a dry ice-ethanol bath and store at -80°C.

We describe experiments using two sources here: a dicot (canola) and a monocot (banana, *Musa acuminata* cv. Rasthali). Each vector directs gene repair of the kanamycin mutation (Table 4); however, the level of correction is elevated 2-3 fold relative to the frequency observed with the chimeric oligonucleotide. These results are similar to those observed in the mammalian system wherein a significant improvement in gene repair occurred when modified single-stranded molecules were used.

Tables are attached hereto.

Table I

Gene repair activity is directed by single-stranded oligonucleotides.

Oligonucleotide	Plasmid	Extract (ug)	kan^r colonies	Fold increase
I	pK^{S}m4021	10	300	
I		20	418	1.0x
II		10	537	
II		20	748	1.78x
III		10	3	
III		20	5	0.01x
IV		10	112	
IV		20	96	0.22x
V		10	217	
V		20	342	0.81x
VI		10	6	
VI		20	39	0.093x
VII		10	0	
VII		20	0	0x
VIII		10	3	
VIII		20	5	0.01x
IX		10	936	
IX		20	1295	3.09x
X		10	1140	
X		20	1588	3.7x
XI		10	480	
XI		20	681	1.6x
XII		10	18	
XII		20	25	0.059x
XIII		10	0	
XIII		20	4	0.009x
-		20	0	
I		-	0	

Plasmid pK^{s}m4021 (1μg), the indicated oligonucleotide (1.5 μg chimeric oligonucleotide or 0.55 μg single-stranded oligonucleotide; molar ratio of oligo to plasmid of 360 to 1) and either 10 or 20 μg of HUH7 cell-free extract were incubated 45 min at 37°C. Isolated plasmid DNA was electroporated into *E. coli* (strain DH10B) and the number of kan^r colonies counted. The data represent the number of kanamycin resistant colonies per 10^6 ampicillin resistant colonies generated from the same reaction and is the average of three

experiments (standard deviation usually less than +/- 15%). Fold increase is defined relative to 418 kan^r colonies (second reaction) and in all reactions was calculated using the 20μg sample.

Table II

Modified single-stranded oligomers are not dependent on MSH2 or MSH3 for optimal gene repair activity.

A. Oligonucleotide	Plasmid	Extract	kan^r colonies
IX (3S/25G)		HUH7	637
X (6S/25G)		HUH7	836
IX		$MEF2^{-/-}$	781
X		$MEF2^{-/-}$	676
IX		$MEF3^{-/-}$	582
X		$MEF3^{-/-}$	530
IX		$MEF^{+/+}$	332
X		$MEF^{+/+}$	497
-		$MEF2^{-/-}$	10
-		$MEF3^{-/-}$	5
-		$MEF^{+/+}$	14

Chimeric oligonucleotide (1.5 μg) or modified single-stranded oligonucleotide (0.55 μg) was incubated with 1μg of plasmid pK^sm4021 and 20μg of the indicated extracts. MEF represents mouse embryonic fibroblasts with either MSH2 ($2^{-/-}$) or MSH3 ($3^{-/-}$) deleted. $MEF^{+/+}$ indicates wild-type mouse embryonic fibroblasts. The other reaction components were then added and processed through the bacterial readout system. The data represent the number of kanamycin resistant colonies per 10^6 ampicillin resistant colonies.

Table III

Frameshift mutation repair is directed by single-stranded oligonucleotides

Oligonucleotide	Plasmid	Extract	tet^r colonies
Tet IX (3S/25A; 0.5 μg)	$pT^s\Delta208$ (1μg)	-	0
-	↓	20μg	0
Tet IX (0.5 μg)	↓	↓	48
Tet IX (1.5 μg)	↓	↓	130
Tet IX (2.0 μg)	↓	↓	68
Tet I (chimera; 1.5 μg)	↓	↓	48

Each reaction mixture contained the indicated amounts of plasmid and oligonucleotide. The extract used for these experiments came from HUH7 cells. The data represent the number of tetracycline resistant colonies per 10^6 ampicillin resistant colonies generated from the same reaction and is the average of 3 independent experiments. Tet I is a chimeric oligonucleotide and Tet IX is a modified single-stranded oligonucleotide that are designed to insert a T residue at position 208 of $pT^s\Delta208$. These oligonucleotides are equivalent to structures I and IX in Figure 2.

Table IV

Plant cell-free extracts support gene repair by single-stranded oligonucleotides

Oligonucleotide	Plasmid	Extract	kan^r colonies
II (chimera)	pK^{S}m4021	30µg Canola	337
IX (3S/25G)		Canola	763
X (6S/25G)		Canola	882
II		*Musa*	203
IX		*Musa*	343
X		*Musa*	746
-		Canola	0
-		*Musa*	0
IX		- Canola	0
X		- *Musa*	0

Canola or Musa cell-free extracts were tested for gene repair activity on the kanamycin-sensitive gene as previously described in (18). Chimeric oligonucleotide II (1.5 µg) and modified single-stranded oligonucleotides IX and X (0.55µg) were used to correct pK^{S}m4021. Total number of kan^r colonies are present per 10^7 ampicillin resistant colonies and represent an average of four independent experiments.

Table V

Gene repair activity in cell-free extracts prepared from yeast (Saccharomyces cerevisiae)

Cell-type	Plasmid	Chimeric Oligo	SS Oligo	$kan^r/amp^r \times 10^6$
Wild type	pKan*m4021	1μg		0.36
Wild type			1μg	0.81
ΔRAD52		1μg		10.72
ΔRAD52			1μg	17.41
ΔPMS1		1μg		2.02
ΔPMS1			1μg	3.23

In this experiment, the kan* gene in pKan*4021 is corrected by either a chimeric double-hairpin oligonucleotide or a single-stranded oligonucleotide containing three thioate linkages at each end (3S/25G).

EXAMPLE 2
Yeast Cell Targeting Assay Method for Base Alteration and Preferred Oligonucleotide Selection

In this example, single-stranded oligonucleotides with modified backbones and double-hairpin oligonucleotides with chimeric, RNA-DNA backbones are used to measure gene repair using two episomal targets with a fusion between a hygromycin resistance gene and eGFP as a target for gene repair. These plasmids are pAURHYG(rep)GFP, which contains a point mutation in the hygromycin resistance gene (Figure 7), pAURHYG(ins)GFP, which contains a single-base insertion in the hygromycin resistance gene (Figure 7) and pAURHYG(Δ)GFP which has a single base deletion. We also use the plasmid containing a wild-type copy of the hygromycin-eGFP fusion gene, designated pAURHYG(wt)GFP, as a control. These plasmids also contain an aureobasidinA resistance gene. In pAURHYG(rep)GFP, hygromycin resistance gene function and green fluorescence from the eGFP protein are restored when a G at position 137, at codon 46 of the hygromycin B coding sequence, is converted to a C thus removing a premature stop codon in the hygromycin resistance gene coding region. In pAURHYG(ins)GFP, hygromycin resistance gene function and green fluorescence from the eGFP protein are restored when an A inserted between nucleotide positions 136 and 137, at codon 46 of the hygromycin B coding sequence, is deleted and a C is substituted for the T at position 137, thus correcting a frameshift mutation and restoring the reading frame of the hygromycin-eGFP fusion gene.

We synthesize the set of three yeast expression constructs pAURHYG(rep)eGFP, pAURHYG(Δ)eGFP, pAURHYG(ins)eGFP, that contain a point mutation at nucleotide 137 of the hygromycin-B coding sequence as follows. (rep) indicates a T137⇨G replacement, (Δ) represents a deletion of the G137 and (ins) represents an A insertion between nucleotides 136 and 137. We construct this set of plasmids by excising the respective expression cassettes by restriction digest from pHyg(x)EGFP and ligation into pAUR123 (Panvera, CA). We digest 10 µg pAUR123 vector DNA, as well as, 10 µg of each pHyg(x)EGFP construct with KpnI and SalI (NEB). We gel purify each of the DNA fragments and prepare them for enzymatic ligation. We ligate each mutated insert into pHygEGFP vector at 3:1 molar ration using T4 DNA ligase (Roche). We screen clones by restriction digest, confirm by Sanger dideoxy chain termination sequencing and purify using a Qiagen maxiprep kit.

We use this system to assay the ability of five oligonucleotides (shown in Figure 8) to support correction under a variety of conditions. The oligonucleotides which direct correction of the mutation in pAURHYG(rep)GFP can also direct correction of the mutation in pAURHYG(ins)GFP. Three of the four oligonucleotides (HygE3T/25, HygE3T/74 and HygGG/Rev) share the same 25-base sequence surrounding the base targeted for alteration. HygGG/Rev is an RNA-DNA chimeric double hairpin

oligonucleotide of the type described in the prior art. One of these oligonucleotides, HygE3T/74, is a 74-base oligonucleotide with the 25-base sequence centrally positioned. The fourth oligonucleotide, designated HygE3T/74α, is the reverse complement of HygE3T/74. The fifth oligonucleotide, designated Kan70T, is a non-specific, control oligonucleotide which is not complementary to the target sequence. Alternatively, an oligonucleotide of identical sequence but lacking a mismatch to the target or a completely thioate modified oligonucleotide or a completely 2-0-methylated modified oligonucleotide may be used as a control.

Oligonucleotide synthesis and cells. We synthesized and purified the chimeric, double-hairpin oligonucleotides and single-stranded oligonucleotides (including those with the indicated modifications) as described in Example 1. Plasmids used for assay were maintained stably in yeast (*Saccharomyces cerevisiae*) strain LSY678 *MATα* at low copy number under aureobasidin selection. Plasmids and oligonucleotides are introduced into yeast cells by electroporation as follows: to prepare electrocompetent yeast cells, we inoculate 10 ml of YPD media from a single colony and grow the cultures overnight with shaking at 300 rpm at 30°C. We then add 30 ml of fresh YPD media to the overnight cultures and continue shaking at 30°C until the OD_{600} was between 0.5 and 1.0 (3-5 hours). We then wash the cells by centrifuging at 4°C at 3000 rpm for 5 minutes and twice resuspending the cells in 25 ml ice-cold distilled water. We then centrifuge at 4°C at 3000 rpm for 5 minutes and resuspend in 1 ml ice-cold 1M sorbitol and then finally centrifuge the cells at 4°C at 5000 rpm for 5 minutes and resuspend the cells in 120 µl 1M sorbitol. To transform electrocompetent cells with plasmids or oligonucleotides, we mix 40 µl of cells with 5 µg of nucleic acid, unless otherwise stated, and incubate on ice for 5 minutes. We then transfer the mixture to a 0.2 cm electroporation cuvette and electroporate with a BIO-RAD Gene Pulser apparatus at 1.5 kV, 25 µF, 200 Ω for one five-second pulse. We then immediately resuspend the cells in 1 ml YPD supplemented with 1M sorbitol and incubate the cultures at 30°C with shaking at 300 rpm for 6 hours. We then spread 200 µl of this culture on selective plates containing 300 µg/ml hygromycin and spread 200 µl of a 10^5 dilution of this culture on selective plates containing 500 ng/ml aureobasidinA and/or and incubate at 30°C for 3 days to allow individual yeast colonies to grow. We then count the colonies on the plates and calculate the gene conversion efficiency by determining the number of hygromycin resistance colonies per 10^5 aureobasidinA resistant colonies.

Frameshift mutations are repaired in yeast cells. We test the ability of the oligonucleotides shown in Figure 8 to correct a frameshift mutation *in vivo* using LSY678 yeast cells containing the plasmid pAURHYG(ins)GFP. These experiments, presented in Table 6, indicate that these oligonucleotides can support gene correction in yeast cells. These data reinforce the results described in

Example 1 indicating that oligonucleotides comprising phosphorothioate linkages facilitate gene correction much more efficiently than control duplex, chimeric RNA-DNA oligonucleotides. This gene correction activity is also specific as transformation of cells with the control oligonucleotide Kan70T produced no hygromycin resistant colonies above background and thus Kan70T did not support gene correction in this system. In addition, we observe that the 74-base oligonucleotide (HygE3T/74) corrects the mutation in pAURHYG(ins)GFP approximately five-fold more efficiently than the 25-base oligonucleotide (HygE3T/25). We also perform control experiments with LSY678 yeast cells containing the plasmid pAURHYG(wt)GFP. With this strain we observed that even without added oligonucleotides, there are too many hygromycin resistant colonies to count.

We also use additional oligonucleotides to assay the ability of individual oligonucleotides to correct multiple mutations in the pAURHYG(x)eGFP plasmid. These include, for example, one that alters two basepairs that are 3 nucleotides apart is a 74-mer with the sequence 5'-CTCGTGCTTTCAGCTTCGATGTAGGAGGGCGTGG**G**TA**C**GTCCTGCGGGTAAATAGCTGCGCCGATG GTTTCTAC-3'; a 74-mer that alters two basepairs that are 15 nucleotides apart with the sequence 5'-CTCGTGCTTTCAGCTTCGATGTAGGAGGGCGTGGATA**C**GTCCTGCGGGTAAA**C**AGCTGCGCCGATG GTTTCTAC-3'; and a 74-mer that alters two basepairs that are 27 nucleotides apart with the sequence 5'-CTCGTGCTTTCAGCTTCGATGTAGGAGGGCGTGGATA**C**GTCCTGCGGGTAAATAGCTGCGCCGA**C**G GTTTCTAC. The nucleotides in these oligonucleotides that direct alteration of the target sequence are underlined and in boldface. These oligonucleotides are modified in the same ways as the other oligonucleotides of the invention.

Oligonucleotides targeting the sense strand direct gene correction more efficiently. We compare the ability of single-stranded oligonucleotides to target each of the two strands of the target sequence of both pAURHYG(ins)GFP and pAURHYG(rep)GFP. These experiments, presented in Tables 7 and 8, indicate that an oligonucleotide, HygE3T/74α, with sequence complementary to the sense strand (i.e. the strand of the target sequence that is identical to the mRNA) of the target sequence facilitates gene correction approximately ten-fold more efficiently than an oligonucleotide, HygE3T/74, with sequence complementary to the non-transcribed strand which serves as the template for the synthesis of RNA. As indicated in Table 7, this effect was observed over a range of oligonucleotide concentrations from 0-3.6 µg, although we did observe some variability in the difference between the two oligonucleotides (indicated in Table 7 as a fold difference between HygE3T/74α and HygE3T/74). Furthermore, as shown in Table 8, we observe increased efficiency of correction by HygE3T/74α relative to HygE3T/74 regardless of whether the oligonucleotides were used to correct the base substitution

mutation in pAURHYG(rep)GFP or the insertion mutation in pAURHYG(ins)GFP. The data presented in Table 8 further indicate that the single-stranded oligonucleotides correct a base substitution mutation more efficiently than an insertion mutation. However, this last effect was much less pronounced and the oligonucleotides of the invention are clearly able efficiently to correct both types of mutations in yeast cells. In addition, the role of transcription is investigated using plasmids with inducible promoters such as that described in Figure 10.

Optimization of oligonucleotide concentration. To determine the optimal concentration of oligonucleotide for the purpose of gene alteration, we test the ability of increasing concentrations of Hyg3T/74α to correct the mutation in pAURHYG(rep)GFP contained in yeast LSY678. We chose this assay system because our previous experiments indicated that it supports the highest level of correction. However, this same approach could be used to determine the optimal concentration of any given oligonucleotide. We test the ability of Hyg3T/74α to correct the mutation in pAURHYG(rep)GFP contained in yeast LSY678 over a range of oligonucleotide concentrations from 0-10.0 μg. As shown in Table 9, we observe that the correction efficiency initially increases with increasing oligonucleotide concentration, but then declines at the highest concentration tested.

Tables are attached hereto.

Table 6

Correction of an insertion mutation in pAURHYG(ins)GFP by HygGG/Rev, HygE3T/25 and HygE3T/74

Oligonucleotide Tested	Colonies on Hygromycin	Colonies on Aureobasidin (/10^5)	Correction Efficiency
HygGG/Rev	3	157	0.02
HygE3T/25	64	147	0.44
HygE3T/74	280	174	1.61
Kan70T	0	—	—

Table 7

An oligonucleotide targeting the sense strand of the target sequence corrects more efficiently.

Amount of Oligonucleotide (μg)	Colonies per hygromycin plate	
	HygE3T/74	HygE3T/74α
0	0	0
0.6	24	128 (8.4x)*
1.2	69	140 (7.5x)*
2.4	62	167 (3.8x)*
3.6	29	367 (15x)*

* The numbers in parentheses represent the fold increase in efficiency for targeting the non-transcribed strand as compared to the other strand of a DNA duplex that encodes a protein.

Table 8

Correction of a base substitution mutation is more efficient than correction of a frame shift mutation.

Oligonucleotide Tested (5 µg)	Plasmid tested (contained in LSY678)	
	pAURHYG(ins)GFP	pAURHYG(rep)GFP
HygE3T/74	72	277
HygE3T/74α	1464	2248
Kan70T	0	0

Table 9

Optimization of oligonucleotide concentration in electroporated yeast cells.

Amount (µg)	Colonies on hygromycin	Colonies on aureobasidin (/10^5)	Correction efficiency
0	0	67	0
1.0	5	64	0.08
2.5	47	30	1.57
5.0	199	33	6.08
7.5	383	39	9.79
10.0	191	33	5.79

Example 3
Cultured Cell Manipulation

Mononuclear cells are isolated from human umbilical cord blood of normal donors using Ficoll Hypaque (Pharmacia Biotech, Uppsala, Sweden) density centrifugation. CD34+ cells are immunomagnetically purified from mononuclear cells using either the progenitor or Multisort Kits (Miltenyi Biotec, Auburn, CA). Lin^-CD38^- cells are purified from the mononuclear cells using negative selection with StemSep system according to the manufacturer's protocol (Stem Cell Technologies, Vancouver, CA).

Cells used for microinjection are either freshly isolated or cryopreserved and cultured in Stem Medium (S Medium) for 2 to 5 days prior to microinjection. S Medium contains Iscoves' Modified Dulbecco's Medium without phenol red (IMDM) with 100 µg/ml glutamine/penicillin/streptomycin, 50 mg/ml bovine serum albumin, 50 µg/ml bovine pancreatic insulin, 1 mg/ml human transferrin, and IMDM; Stem Cell Technologies), 40 µg/ml low-density lipoprotein (LDL; Sigma, St. Louis, MO), 50 mM HEPEs buffer and 50 µM 2-mercaptoethanol, 20 ng/ml each of thrombopoietin, flt-3 ligand, stem cell factor and human IL-6 (Pepro Tech Inc., Rocky Hill, NJ). After microinjection, cells are detached and transferred in bulk into wells of 48 well plates for culturing.

35 mm dishes are coated overnight at 4° C with 50 µg/ml Fibronectin (FN) fragment CH-296 (Retronectin; TaKaRa Biomedicals, Panvera, Madison, WI) in phosphate buffered saline and washed with IMDM containing glutamine/penicillin/streptomycin. 300 to 2000 cells are added to cloning rings and attached to the plates for 45 minutes at 37° C prior to microinjection. After incubation, cloning rings are removed and 2 ml of S Medium are added to each dish for microinjection. Pulled injection needles with a range of 0.22 µ to 0.3 µ outer tip diameter are used. Cells are visualized with a microscope equipped with a temperature controlled stage set at 37° C and injected using an electronically interfaced Eppendorf Micromanipulator and Transjector. Successfully injected cells are intact, alive and remain attached to the plate post injection. Molecules that are flourescently labeled allow determination of the amount of oligonucleotide delivered to the cells.

For in vitro erythropoiesis from Lin^-CD38^- cells, the procedure of Malik, 1998 can be used. Cells are cultured in ME Medium for 4 days and then cultured in E Medium for 3 weeks. Erythropoiesis is evident by glycophorin A expression as well as the presence of red color representing the presence of hemoglobin in the cultured cells. The injected cells are able to retain their proliferative capacity and the ability to generate myeloid and erythoid progeny. CD34+ cells can convert a normal A (β^A) to sickle T (β^S) mutation in the β-globin gene or can be altered using any of the oligonucleotides of the invention herein for correction or alteration of a normal gene to a mutant gene. Alternatively, stem cells can be isolated from blood of humans having genetic disease mutations and the oligonucleotides of the invention can be used to correct a defect or to modify genomes within those cells.

Alternatively, non-stem cell populations of cultured cells can be manipulated using any method known to those of skill in the art including, for example, the use of polycations, cationic lipids, liposomes, polyethylenimine (PEI), electroporation, biolistics, calcium phophate precipitation, or any other method known in the art.

Notes on the tables presented below:

Each of the following tables presents, for the specified human gene, a plurality of mutations that are known to confer a clinically-relevant phenotype and, for each mutation, the oligonucleotides that can be used to correct the respective mutation site-specifically in the human genome according to the present invention.

The left-most column identifies each mutation and the clinical phenotype that the mutation confers.

For most entries, the mutation is identified at both the nucleic acid and protein level. At the amino acid level, mutations are presented according to the following standard nomenclature. The centered number identifies the position of the mutated codon in the protein sequence; to the left of the number is the wild type residue and to the right of the number is the mutant codon. Codon numbering is according to the Human Gene Mutation Database, Cardiff, Wales, UK (http://archive.uwcm.ac.uk/search/mg/allgenes). Terminator codons are shown as "TERM". At the nucleic acid level, the entire triplet of the wild type and mutated codons is shown.

The middle column presents, for each mutation, four oligonucleotides capable of repairing the mutation site-specifically in the human genome or in cloned human DNA including human DNA in artificial chromosomes, episomes, plasmids, or other types of vectors. The oligonucleotides of the invention, however, may include any of the oligonucleotides sharing portions of the sequence of the 121 base sequence. Thus, oligonucleotides of the invention for each of the depicted targets may be 18, 19, 20 up to about 121 nucleotides in length. Sequence may be added non-symmetrically.

All oligonucleotides are presented, per convention, in the 5' to 3' orientation. The nucleotide that effects the change in the genome is underlined and presented in bold.

The first of the four oligonucleotides for each mutation is a 121 nt oligonucleotide centered about the repair nucleotide. The second oligonucleotide, its reverse complement, targets the opposite strand of the DNA duplex for repair. The third oligonucleotide is the minimal 17 nt domain of the first oligonucleotide, also centered about the repair nucleotide. The fourth oligonucleotide is the reverse complement of the third, and thus represents the minimal 17 nt domain of the second.

The third column of each table presents the SEQ ID NO: of the respective repair oligonucleotide.

EXAMPLE 4

Adenosine Deaminase (ADA)

Adenosine deaminase (ADA, EC 3.5.4.4) catalyses the deamination of adenosine and 2'-deoxyadenosine to inosine or 2'-deoxyinosine respectively. ADA deficiency has been identified as the metabolic basis for 20-30% of cases with recessively inherited severe combined immunodeficiency (SCID). Affected infants are subject to recurrent chronic viral, fungal, protozoal, and bacterial infections and frequently present with persistent diarrhea, failure to thrive and candidiasis. In patients homozygous for ADA deficiency, 2'-deoxyadenosine accumulating during the rapid turnover of cells rich in DNA is converted back to dATP, either by adenosine kinase or deoxycytidine kinase. Many hypotheses have been advanced to explain the specific toxicity to the immune system in ADA deficiency. The apparently selective accumulation of dATP in thymocytes and peripheral blood B cells, with resultant inhibition of ribonucleotide reductase and DNA synthesis is probably the principal mechanism.

The structural gene for ADA is encoded as a single 32 kb locus containing 12 exons. Studies of the molecular defect in ADA-deficient patients have shown that mRNA is usually detectable in normal or supranormal amounts. Specific base substitution mutations have been detected in the majority of cases with the complete deficiency. A C-to-T base substitution mutation in exon 11 accounts for a high proportion of these, whilst a few patients are homozygous for large deletions encompassing exon I. A common point mutation resulting in a heat-labile ADA has been characterised in some patients with partial ADA deficiency, a disorder with an apparently increased prevalence in the Caribbean.

As yet no totally effective therapy for ADA deficiency has been reported, except in those few cases where bone marrow from an HLA/MLR compatible sibling donor was available.

Two therapeutic approaches have provided long-term benefit in specific instances. First, reconstitution using T cell depleted mismatched sibling marrow has been encouraging, particularly in early presenters completely deficient in ADA. Secondly, therapy with polyethylene glycol-modified adenosine deaminase (PEG-ADA) for more than 5 years has produced a sustained increase in lymphocyte numbers and mitogen responses together with evidence of in vivo B cell function. Success has generally been achieved in late presenters with residual ADA activity in mononuclear cells.

ADA deficiency has been chosen as the candidate disease for gene replacement therapy and the first human experiment commenced in 1990. The clinical consequences of overexpression of ADA activity - one of the potential hazards of gene implant - are known and take the form of an hereditary haemolytic anaemia associated with a tissue-specific increase in ADA activity. The genetic basis for the

latter autosomal dominant disorder seemingly relates to markedly increased levels of structurally normal ADA mRNA.

Table 10
ADA Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency GLN3TERM CAG to TAG	AGAGACCCACCGAGCGGCGGCGGAGGGAGCAGCGCCGGGG CGCACGAGGGCACCATGGCC**C**AGACGCCCGCCTTCGACAAG CCCAAAGTGAGCGCGCGCGGGGGCTCCGGGGACGGGGGTC	1
	GACCCCCGTCCCCGGAGCCCCCGCGCGCGCTCACTTTGGG CTTGTCGAAGGCGGGCGTCT**G**GGCCATGGTGCCCTCGTGCG CCCCGGCGCTGCTCCCTCCGCCGCCGCTCGGTGGGTCTCT	2
	CCATGGCC**C**AGACGCCC	3
	GGGCGTCT**G**GGCCATGG	4
Adenosine deaminase deficiency HIS15ASP CAT to GAT	TATTTGTTCTCTCTCTCCCTTTCTCTCTCTCTTCCCCCTGCCC CCTTGCAGGTAGAACTG**C**ATGTCCACCTAGACGGATCCATCA AGCCTGAAACCATCTTATACTATGGCAGGTAAGTCC	5
	GGACTTACCTGCCATAGTATAAGATGGTTTCAGGCTTGATGGA TCCGTCTAGGTGGACAT**G**CAGTTCTACCTGCAAGGGGGCAG GGGGAAGAGAGAGAGAAAGGGAGAGAGAGAACAAATA	6
	TAGAACTG**C**ATGTCCAC	7
	GTGGACAT**G**CAGTTCTA	8
Adenosine deaminase deficiency GLY20ARG GGA to AGA	TCCCTTTCTCTCTCTCTTCCCCCTGCCCCCTTGCAGGTAGAA CTGCATGTCCACCTAGAC**G**GATCCATCAAGCCTGAAACCATC TTATACTATGGCAGGTAAGTCCATACAGAAGAGCCCT	9
	AGGGCTCTTCTGTATGGACTTACCTGCCATAGTATAAGATGGT TTCAGGCTTGATGGATC**C**GTCTAGGTGGACATGCAGTTCTAC CTGCAAGGGGGCAGGGGGAAGAGAGAGAGAAAGGGA	10
	ACCTAGAC**G**GATCCATC	11
	GATGGATC**C**GTCTAGGT	12
Adenosine deaminase deficiency GLY74CYS GGC to TGC	CCTGGAGCTCCCAAGGGACTTGGGGAAGGTTGTTCCCAACC CCTTTCTTCCCTTCCCAGG**G**GCTGCCGGGAGGCTATCAAAAG GATCGCCTATGAGTTTGTAGAGATGAAGGCCAAAGAGG	13

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCTCTTTGGCCTTCATCTCTACAAACTCATAGGCGATCCTTTT GATAGCCTCCCGGCAGCCCCTGGGAAGGGAAGAAAGGGGTT GGGAACAACCTTCCCCAAGTCCCTTGGGAGCTCCAGG	14
	CTATCGCGGGCTGCCGG	15
	CCGGCAGCCCGCGATAG	16
Adenosine Deaminase Deficiency ARG76TRP CGG to TGG	GCTCCCAAGGGACTTGGGGAAGGTTGTTCCCAACCCCTTTCT TCCCTTCCCAGGGGCTGCCGGGAGGCTATCAAAAGGATCGC CTATGAGTTTGTAGAGATGAAGGCCAAAGAGGGCGTGG	17
	CCACGCCCTCTTTGGCCTTCATCTCTACAAACTCATAGGCGAT CCTTTTGATAGCCTCCCGGCAGCCCCTGGGAAGGGAAGAAA GGGGTTGGGAACAACCTTCCCCAAGTCCCTTGGGAGC	18
	GGGGCTGCCGGGAGGCT	19
	AGCCTCCCGGCAGCCCC	20
Adenosine Deaminase Deficiency LYS80ARG AAA to AGA	TTGGGGAAGGTTGTTCCCAACCCCTTTCTTCCCTTCCCAGGG GCTGCCGGGAGGCTATCAAAAGGATCGCCTATGAGTTTGTAG AGATGAAGGCCAAAGAGGGCGTGGTGTATGTGGAGGT	21
	ACCTCCACATACACCACGCCCTCTTTGGCCTTCATCTCTACAA ACTCATAGGCGATCCTTTTGATAGCCTCCCGGCAGCCCCTGG GAAGGGAAGAAAGGGGTTGGGAACAACCTTCCCCAA	22
	GGCTATCAAAAGGATCG	23
	CGATCCTTTTGATAGCC	24
Adenosine deaminase deficiency ALA83ASP GCC to GAC	GTTGTTCCCAACCCCTTTCTTCCCTTCCCAGGGGCTGCCGGG AGGCTATCAAAAGGATCGCCTATGAGTTTGTAGAGATGAAGG CCAAAGAGGGCGTGGTGTATGTGGAGGTGCGGTACAG	25
	CTGTACCGCACCTCCACATACACCACGCCCTCTTTGGCCTTC ATCTCTACAAACTCATAGGCGATCCTTTTGATAGCCTCCCGGC AGCCCCTGGGAAGGGAAGAAAGGGGTTGGGAACAAC	26
	AAGGATCGCCTATGAGT	27
	ACTCATAGGCGATCCTT	28
Adenosine deaminase deficiency TYR97CYS TAT to TGT	AGGCTATCAAAAGGATCGCCTATGAGTTTGTAGAGATGAAGG CCAAAGAGGGCGTGGTGTATGTGGAGGTGCGGTACAGTCCG CACCTGCTGGCCAACTCCAAAGTGGAGCCAATCCCCTG	29
	CAGGGGATTGGCTCCACTTTGGAGTTGGCCAGCAGGTGCGG ACTGTACCGCACCTCCACATACACCACGCCCTCTTTGGCCTT CATCTCTACAAACTCATAGGCGATCCTTTTGATAGCCT	30

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CGTGGTGTATGTGGAGG	31
	CCTCCACATACACCACG	32
Adenosine deaminase deficiency ARG101GLN CGG to CAG	GGATCGCCTATGAGTTTGTAGAGATGAAGGCCAAAGAGGGCG TGGTGTATGTGGAGGTGCGGTACAGTCCGCACCTGCTGGCC AACTCCAAAGTGGAGCCAATCCCCTGGAACCAGGCTGA	33
	TCAGCCTGGTTCCAGGGGATTGGCTCCACTTTGGAGTTGGCC AGCAGGTGCGGACTGTACCGCACCTCCACATACACCACGCC CTCTTTGGCCTTCATCTCTACAAACTCATAGGCGATCC	34
	GGAGGTGCGGTACAGTC	35
	GACTGTACCGCACCTCC	36
Adenosine deaminase deficiency ARG101LEU CGG to CTG	GGATCGCCTATGAGTTTGTAGAGATGAAGGCCAAAGAGGGCG TGGTGTATGTGGAGGTGCGGTACAGTCCGCACCTGCTGGCC AACTCCAAAGTGGAGCCAATCCCCTGGAACCAGGCTGA	37
	TCAGCCTGGTTCCAGGGGATTGGCTCCACTTTGGAGTTGGCC AGCAGGTGCGGACTGTACCGCACCTCCACATACACCACGCC CTCTTTGGCCTTCATCTCTACAAACTCATAGGCGATCC	38
	GGAGGTGCGGTACAGTC	39
	GACTGTACCGCACCTCC	40
Adenosine deaminase deficiency ARG101TRP CGG to TGG	AGGATCGCCTATGAGTTTGTAGAGATGAAGGCCAAAGAGGGC GTGGTGTATGTGGAGGTGCGGTACAGTCCGCACCTGCTGGC CAACTCCAAAGTGGAGCCAATCCCCTGGAACCAGGCTG	41
	CAGCCTGGTTCCAGGGGATTGGCTCCACTTTGGAGTTGGCCA GCAGGTGCGGACTGTACCGCACCTCCACATACACCACGCCC TCTTTGGCCTTCATCTCTACAAACTCATAGGCGATCCT	42
	TGGAGGTGCGGTACAGT	43
	ACTGTACCGCACCTCCA	44
Adenosine deaminase deficiency PRO104LEU CCG to CTG	ATGAGTTTGTAGAGATGAAGGCCAAAGAGGGCGTGGTGTATG TGGAGGTGCGGTACAGTCCGCACCTGCTGGCCAACTCCAAA GTGGAGCCAATCCCCTGGAACCAGGCTGAGTGAGTGAT	45
	ATCACTCACTCAGCCTGGTTCCAGGGGATTGGCTCCACTTTG GAGTTGGCCAGCAGGTGCGGACTGTACCGCACCTCCACATA CACCACGCCCTCTTTGGCCTTCATCTCTACAAACTCAT	46
	GTACAGTCCGCACCTGC	47
	GCAGGTGCGGACTGTAC	48

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency LEU106VAL CTG to GTG	TTTGTAGAGATGAAGGCCAAAGAGGGCGTGGTGTATGTGGAG GTGCGGTACAGTCCGCACCTGCTGGCCAACTCCAAAGTGGA GCCAATCCCCTGGAACCAGGCTGAGTGAGTGATGGGCC	49
	GGCCCATCACTCACTCAGCCTGGTTCCAGGGGATTGGCTCCA CTTTGGAGTTGGCCAGCAGGTGCGGACTGTACCGCACCTCC ACATACACCACGCCCTCTTTGGCCTTCATCTCTACAAA	50
	GTCCGCACCTGCTGGCC	51
	GGCCAGCAGGTGCGGAC	52
Adenosine deaminase deficiency LEU107PRO CTG to CCG	TAGAGATGAAGGCCAAAGAGGGCGTGGTGTATGTGGAGGTG CGGTACAGTCCGCACCTGCTGGCCAACTCCAAAGTGGAGCC AATCCCCTGGAACCAGGCTGAGTGAGTGATGGGCCTGGA	53
	TCCAGGCCCATCACTCACTCAGCCTGGTTCCAGGGGATTGGC TCCACTTTGGAGTTGGCCAGCAGGTGCGGACTGTACCGCAC CTCCACATACACCACGCCCTCTTTGGCCTTCATCTCTA	54
	GCACCTGCTGGCCAACT	55
	AGTTGGCCAGCAGGTGC	56
Adenosine deaminase deficiency PRO126GLN CCA to CAA	GCCTTCCTTTTGCCTCAGGCCCATCCCTACTCCTCTCCTCAC ACAGAGGGGACCTCACCCCAGACGAGGTGGTGGCCCTAGTG GGCCAGGGCCTGCAGGAGGGGGAGCGAGACTTCGGGGT	57
	ACCCCGAAGTCTCGCTCCCCCTCCTGCAGGCCCTGGCCCAC TAGGGCCACCACCTCGTCTGGGGTGAGGTCCCCTCTGTGTG AGGAGAGGAGTAGGGATGGGCCTGAGGCAAAAGGAAGGC	58
	CCTCACCCCAGACGAGG	59
	CCTCGTCTGGGGTGAGG	60
Adenosine deaminase deficiency VAL129MET GTG to ATG	TTTGCCTCAGGCCCATCCCTACTCCTCTCCTCACACAGAGGG GACCTCACCCCAGACGAGGTGGTGGCCCTAGTGGGCCAGGG CCTGCAGGAGGGGGAGCGAGACTTCGGGGTCAAGGCCC	61
	GGGCCTTGACCCCGAAGTCTCGCTCCCCCTCCTGCAGGCCC TGGCCCACTAGGGCCACCACCTCGTCTGGGGTGAGGTCCCC TCTGTGTGAGGAGAGGAGTAGGGATGGGCCTGAGGCAAA	62
	CAGACGAGGTGGTGGCC	63
	GGCCACCACCTCGTCTG	64

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency GLY140GLU GGG to GAG	ACAGAGGGGACCTCACCCCAGACGAGGTGGTGGCCCTAGTG GGCCAGGGCCTGCAGGAGG**G**GGAGCGAGACTTCGGGGTCA AGGCCCGGTCCATCCTGTGCTGCATGCGCCACCAGCCCAG	65
	CTGGGCTGGTGGCGCATGCAGCACAGGATGGACCGGGCCTT GACCCCGAAGTCTCGCTCC**C**CCTCCTGCAGGCCCTGGCCCA CTAGGGCCACCACCTCGTCTGGGGTGAGGTCCCCTCTGT	66
	GCAGGAGG**G**GGAGCGAG	67
	CTCGCTCC**C**CCTCCTGC	68
Adenosine deaminase deficiency ARG142GLN CGA to CAA	GGGACCTCACCCCAGACGAGGTGGTGGCCCTAGTGGGCCAG GGCCTGCAGGAGGGGGAGC**G**AGACTTCGGGGTCAAGGCCC GGTCCATCCTGTGCTGCATGCGCCACCAGCCCAGTGAGTA	69
	TACTCACTGGGCTGGTGGCGCATGCAGCACAGGATGGACCG GGCCTTGACCCCGAAGTCT**C**GCTCCCCCTCCTGCAGGCCCT GGCCCACTAGGGCCACCACCTCGTCTGGGGTGAGGTCCC	70
	GGGGGAGC**G**AGACTTCG	71
	CGAAGTCT**C**GCTCCCCC	72
Adenosine deaminase deficiency ARG142TERM CGA to TGA	GGGGACCTCACCCCAGACGAGGTGGTGGCCCTAGTGGGCCA GGGCCTGCAGGAGGGGGAG**C**GAGACTTCGGGGTCAAGGCC CGGTCCATCCTGTGCTGCATGCGCCACCAGCCCAGTGAGT	73
	ACTCACTGGGCTGGTGGCGCATGCAGCACAGGATGGACCGG GCCTTGACCCCGAAGTCTC**G**CTCCCCCTCCTGCAGGCCCTG GCCCACTAGGGCCACCACCTCGTCTGGGGTGAGGTCCCC	74
	AGGGGGAG**C**GAGACTTC	75
	GAAGTCTC**G**CTCCCCCT	76
Adenosine deaminase deficiency ARG149GLN CGG to CAG	TGGTGGCCCTAGTGGGCCAGGGCCTGCAGGAGGGGGAGCG AGACTTCGGGGTCAAGGCCC**G**GTCCATCCTGTGCTGCATGC GCCACCAGCCCAGTGAGTAGGATCACCGCCCTGCCCAGGG	77
	CCCTGGGCAGGGCGGTGATCCTACTCACTGGGCTGGTGGCG CATGCAGCACAGGATGGAC**C**GGGCCTTGACCCCGAAGTCTC GCTCCCCCTCCTGCAGGCCCTGGCCCACTAGGGCCACCA	78
	CAAGGCCC**G**GTCCATCC	79
	GGATGGAC**C**GGGCCTTG	80

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency ARG149TRP CGG to TGG	GTGGTGGCCCTAGTGGGCCAGGGCCTGCAGGAGGGGGAGC GAGACTTCGGGGTCAAGGCC**C**GGTCCATCCTGTGCTGCATG CGCCACCAGCCCAGTGAGTAGGATCACCGCCCTGCCCAGG	81
	CCTGGGCAGGGCGGTGATCCTACTCACTGGGCTGGTGGCGC ATGCAGCACAGGATGGACC**G**GGCCTTGACCCCGAAGTCTCG CTCCCCCTCCTGCAGGCCCTGGCCCACTAGGGCCACCAC	82
	TCAAGGCC**C**GGTCCATC	83
	GATGGACC**G**GGCCTTGA	84
Adenosine deaminase deficiency LEU152MET CTG to ATG	CTAGTGGGCCAGGGCCTGCAGGAGGGGGAGCGAGACTTCG GGGTCAAGGCCCGGTCCATC**C**TGTGCTGCATGCGCCACCAG CCCAGTGAGTAGGATCACCGCCCTGCCCAGGGCCGCCCGT	85
	ACGGGCGGCCCTGGGCAGGGCGGTGATCCTACTCACTGGG CTGGTGGCGCATGCAGCACA**G**GATGGACCGGGCCTTGACCC CGAAGTCTCGCTCCCCCTCCTGCAGGCCCTGGCCCACTAG	86
	GGTCCATC**C**TGTGCTGC	87
	GCAGCACA**G**GATGGACC	88
Adenosine deaminase deficiency ARG156CYS CGC to TGC	GGCCTGCAGGAGGGGGAGCGAGACTTCGGGGTCAAGGCCC GGTCCATCCTGTGCTGCATG**C**GCCACCAGCCCAGTGAGTAG GATCACCGCCCTGCCCAGGGCCGCCCGTCTCACCCTGGCC	89
	GGCCAGGGTGAGACGGGCGGCCCTGGGCAGGGCGGTGATC CTACTCACTGGGCTGGTGGC**G**CATGCAGCACAGGATGGACC GGGCCTTGACCCCGAAGTCTCGCTCCCCCTCCTGCAGGCC	90
	GCTGCATG**C**GCCACCAG	91
	CTGGTGGC**G**CATGCAGC	92
Adenosine deaminase deficiency ARG156HIS CGC to CAC	GCCTGCAGGAGGGGGAGCGAGACTTCGGGGTCAAGGCCCG GTCCATCCTGTGCTGCATGC**G**CCACCAGCCCAGTGAGTAGG ATCACCGCCCTGCCCAGGGCCGCCCGTCTCACCCTGGCCC	93
	GGGCCAGGGTGAGACGGGCGGCCCTGGGCAGGGCGGTGAT CCTACTCACTGGGCTGGTGG**C**GCATGCAGCACAGGATGGAC CGGGCCTTGACCCCGAAGTCTCGCTCCCCCTCCTGCAGGC	94
	CTGCATGC**G**CCACCAGC	95
	GCTGGTGG**C**GCATGCAG	96

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency VAL177MET GTG to ATG	CTGCCCACAGACTGGTCCCCCAAGGTGGTGGAGCTGTGTAA GAAGTACCAGCAGCAGACCGTGGTAGCCATTGACCTGGCTG GAGATGAGACCATCCCAGGAAGCAGCCTCTTGCCTGGAC	97
	GTCCAGGCAAGAGGCTGCTTCCTGGGATGGTCTCATCTCCAG CCAGGTCAATGGCTACCACGGTCTGCTGCTGGTACTTCTTAC ACAGCTCCACCACCTTGGGGGACCAGTCTGTGGGCAG	98
	AGCAGACCGTGGTAGCC	99
	GGCTACCACGGTCTGCT	100
Adenosine deaminase deficiency ALA179ASP GCC to GAC	CAGACTGGTCCCCCAAGGTGGTGGAGCTGTGTAAGAAGTAC CAGCAGCAGACCGTGGTAGCCATTGACCTGGCTGGAGATGA GACCATCCCAGGAAGCAGCCTCTTGCCTGGACATGTCCA	101
	TGGACATGTCCAGGCAAGAGGCTGCTTCCTGGGATGGTCTCA TCTCCAGCCAGGTCAATGGCTACCACGGTCTGCTGCTGGTAC TTCTTACACAGCTCCACCACCTTGGGGGACCAGTCTG	102
	CGTGGTAGCCATTGACC	103
	GGTCAATGGCTACCACG	104
Adenosine deaminase deficiency GLN199PRO CAG to CCG	CCATTGACCTGGCTGGAGATGAGACCATCCCAGGAAGCAGC CTCTTGCCTGGACATGTCCAGGCCTACCAGGTGGGTCCTGT GAGAAGGAATGGAGAGGCTGGCCCTGGGTGAGCTTGTCT	105
	AGACAAGCTCACCCAGGGCCAGCCTCTCCATTCCTTCTCACA GGACCCACCTGGTAGGCCTGGACATGTCCAGGCAAGAGGCT GCTTCCTGGGATGGTCTCATCTCCAGCCAGGTCAATGG	106
	ACATGTCCAGGCCTACC	107
	GGTAGGCCTGGACATGT	108
Adenosine deaminase deficiency ARG211CYS CGT to TGT	GCTAGGGCACCCATGACCTGGCTCTCCCCCTTCCAGGAGGC TGTGAAGAGCGGCATTCACCGTACTGTCCACGCCGGGGAGG TGGGCTCGGCCGAAGTAGTAAAAGAGGTGAGGGCCTGGG	109
	CCCAGGCCCTCACCTCTTTTACTACTTCGGCCGAGCCCACCT CCCCGGCGTGGACAGTACGGTGAATGCCGCTCTTCACAGCC TCCTGGAAGGGGGAGAGCCAGGTCATGGGTGCCCTAGC	110
	GCATTCACCGTACTGTC	111
	GACAGTACGGTGAATGC	112

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency ARG211HIS CGT to CAT	CTAGGGCACCCATGACCTGGCTCTCCCCCTTCCAGGAGGCT GTGAAGAGCGGCATTCACC**G**TACTGTCCACGCCGGGGAGGT GGGCTCGGCCGAAGTAGTAAAAGAGGTGAGGGCCTGGGC	113
	GCCCAGGCCCTCACCTCTTTTACTACTTCGGCCGAGCCCACC TCCCCGGCGTGGACAGTA**C**GGTGAATGCCGCTCTTCACAGC CTCCTGGAAGGGGGAGAGCCAGGTCATGGGTGCCCTAG	114
	CATTCACC**G**TACTGTCC	115
	GGACAGTA**C**GGTGAATG	116
Adenosine deaminase deficiency ALA215THR GCC to ACC	ATGACCTGGCTCTCCCCCTTCCAGGAGGCTGTGAAGAGCGG CATTCACCGTACTGTCCAC**G**CCGGGGAGGTGGGCTCGGCCG AAGTAGTAAAAGAGGTGAGGGCCTGGGCTGGCCATGGGG	117
	CCCCATGGCCAGCCCAGGCCCTCACCTCTTTTACTACTTCGG CCGAGCCCACCTCCCCGG**C**GTGGACAGTACGGTGAATGCCG CTCTTCACAGCCTCCTGGAAGGGGGAGAGCCAGGTCAT	118
	CTGTCCAC**G**CCGGGGAG	119
	CTCCCCGG**C**GTGGACAG	120
Adenosine deaminase deficiency GLY216ARG GGG to AGG	ACCTGGCTCTCCCCCTTCCAGGAGGCTGTGAAGAGCGGCAT TCACCGTACTGTCCACGCC**G**GGGAGGTGGGCTCGGCCGAAG TAGTAAAAGAGGTGAGGGCCTGGGCTGGCCATGGGGTCC	121
	GGACCCCATGGCCAGCCCAGGCCCTCACCTCTTTTACTACTT CGGCCGAGCCCACCTCCC**C**GGCGTGGACAGTACGGTGAATG CCGCTCTTCACAGCCTCCTGGAAGGGGGAGAGCCAGGT	122
	TCCACGCC**G**GGGAGGTG	123
	CACCTCCC**C**GGCGTGGA	124
Adenosine deaminase deficiency GLU217LYS GAG to AAG	TGGCTCTCCCCCTTCCAGGAGGCTGTGAAGAGCGGCATTCA CCGTACTGTCCACGCCGGG**G**AGGTGGGCTCGGCCGAAGTAG TAAAAGAGGTGAGGGCCTGGGCTGGCCATGGGGTCCCTC	125
	GAGGGACCCCATGGCCAGCCCAGGCCCTCACCTCTTTTACTA CTTCGGCCGAGCCCACCT**C**CCCGGCGTGGACAGTACGGTGA ATGCCGCTCTTCACAGCCTCCTGGAAGGGGGAGAGCCA	126
	ACGCCGGG**G**AGGTGGGC	127
	GCCCACCT**C**CCCGGCGT	128

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency THR233ILE ACA to ATA	CTGCCTCCTCCCATACTTGGCTCTATTCTGCTTCTCTACAGGC TGTGGACATACTCAAGA**C**AGAGCGGCTGGGACACGGCTACC ACACCCTGGAAGACCAGGCCCTTTATAACAGGCTGCG	129
	CGCAGCCTGTTATAAAGGGCCTGGTCTTCCAGGGTGTGGTAG CCGTGTCCCAGCCGCTCT**G**TCTTGAGTATGTCCACAGCCTGT AGAGAAGCAGAATAGAGCCAAGTATGGGAGGAGGCAG	130
	ACTCAAGA**C**AGAGCGGC	131
	GCCGCTCT**G**TCTTGAGT	132
Adenosine deaminase deficiency ARG253PRO CGG to CCG	CAGAGCGGCTGGGACACGGCTACCACACCCTGGAAGACCAG GCCCTTTATAACAGGCTGC**G**GCAGGAAAACATGCACTTCGAG GTAAGCGGGCCAGGGAGTGGGGAGGAACCATCCCCGGC	133
	GCCGGGGATGGTTCCTCCCCACTCCCTGGCCCGCTTACCTC GAAGTGCATGTTTTCCTGC**C**GCAGCCTGTTATAAAGGGCCTG GTCTTCCAGGGTGTGGTAGCCGTGTCCCAGCCGCTCTG	134
	CAGGCTGC**G**GCAGGAAA	135
	TTTCCTGC**C**GCAGCCTG	136
Adenosine deaminase deficiency GLN254TERM CAG to TAG	GAGCGGCTGGGACACGGCTACCACACCCTGGAAGACCAGGC CCTTTATAACAGGCTGCGG**C**AGGAAAACATGCACTTCGAGGT AAGCGGGCCAGGGAGTGGGGAGGAACCATCCCCGGCTG	137
	CAGCCGGGGATGGTTCCTCCCCACTCCCTGGCCCGCTTACC TCGAAGTGCATGTTTTCCT**G**CCGCAGCCTGTTATAAAGGGCC TGGTCTTCCAGGGTGTGGTAGCCGTGTCCCAGCCGCTC	138
	GGCTGCGG**C**AGGAAAAC	139
	GTTTTCCT**G**CCGCAGCC	140
Adenosine deaminase deficiency PRO274LEU CCG to CTG	CCACACACCTGCTCTTCCAGATCTGCCCCTGGTCCAGCTACC TCACTGGTGCCTGGAAGC**C**GGACACGGAGCATGCAGTCATT CGGTGAGCTCTGTTCCCCTGGGCCTGTTCAATTTTGTT	141
	AACAAAATTGAACAGGCCCAGGGGAACAGAGCTCACCGAATG ACTGCATGCTCCGTGTCC**G**GCTTCCAGGCACCAGTGAGGTA GCTGGACCAGGGGCAGATCTGGAAGAGCAGGTGTGTGG	142
	CTGGAAGC**C**GGACACGG	143
	CCGTGTCC**G**GCTTCCAG	144

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency SER291LEU TCG to TTG	GGAGGCTGATTCTCTCCTCCTCCCTCTTCTGCAGGCTCAAAA ATGACCAGGCTAACTACTCGCTCAACACAGATGACCCGCTCA TCTTCAAGTCCACCCTGGACACTGATTACCAGATGAC	145
	GTCATCTGGTAATCAGTGTCCAGGGTGGACTTGAAGATGAGC GGGTCATCTGTGTTGAGCGAGTAGTTAGCCTGGTCATTTTTGA GCCTGCAGAAGAGGGAGGAGGAGAGAATCAGCCTCC	146
	TAACTACTCGCTCAACA	147
	TGTTGAGCGAGTAGTTA	148
Adenosine deaminase deficiency PRO297GLN CCG to CAG	CCTCCCTCTTCTGCAGGCTCAAAAATGACCAGGCTAACTACT CGCTCAACACAGATGACCCGCTCATCTTCAAGTCCACCCTGG ACACTGATTACCAGATGACCAAACGGGACATGGGCTT	149
	AAGCCCATGTCCCGTTTGGTCATCTGGTAATCAGTGTCCAGG GTGGACTTGAAGATGAGCGGGTCATCTGTGTTGAGCGAGTAG TTAGCCTGGTCATTTTTGAGCCTGCAGAAGAGGGAGG	150
	AGATGACCCGCTCATCT	151
	AGATGAGCGGGTCATCT	152
Adenosine deaminase deficiency LEU304ARG CTG to CGG	AAAATGACCAGGCTAACTACTCGCTCAACACAGATGACCCGC TCATCTTCAAGTCCACCCTGGACACTGATTACCAGATGACCAA ACGGGACATGGGCTTTACTGAAGAGGAGTTTAAAAG	153
	CTTTTAAACTCCTCTTCAGTAAAGCCCATGTCCCGTTTGGTCA TCTGGTAATCAGTGTCCAGGGTGGACTTGAAGATGAGCGGGT CATCTGTGTTGAGCGAGTAGTTAGCCTGGTCATTTT	154
	GTCCACCCTGGACACTG	155
	CAGTGTCCAGGGTGGAC	156
Adenosine deaminase deficiency ALA329VAL C-to-T at base 1081	GCCTTCTTTGTTCTCTGGTTCCATGTTGTCTGCCATTCTGGCC TTTCCAGAACATCAATGCGGCCAAATCTAGTTTCCTCCCAGAA GATGAAAAGAGGGAGCTTCTCGACCTGCTCTATAA	157
	TTATAGAGCAGGTCGAGAAGCTCCCTCTTTTCATCTTCTGGGA GGAAACTAGATTTGGCCGCATTGATGTTCTGGAAAGGCCAGA ATGGCAGACAACATGGAACCAGAGAACAAAGAAGGC	158
	CATCAATGCGGCCAAAT	159
	ATTTGGCCGCATTGATG	160

EXAMPLE 5
P53 Mutations

The p53 gene codes for a protein that acts as a transcription factor and serves as a key regulator of the cell cycle. Mutation in this gene is probably the most significant genetic change characterizing the transformation of cells from normalcy to malignancy.

Inactivation of p53 by mutation disrupts the cell cycle which, in turn, sets the stage for tumor formation. Mutations in the p53 gene are among the most commonly diagnosed genetic disorders, occuring in as many as 50% of cancer patients. For some types of cancer, most notably of the breast, lung and colon, p53 mutations are the predominant genetic alternations found thus far. These mutations are associated with genomic instability and thus an increased susceptibility to cancer. Some p53 lesions result in malignancies that are resistant to the most widely used therapeutic regimens and therefore demand more aggressive treatment.

That p53 is associated with different malignant tumors is illustrated in the Li-Fraumeni autosomal dominant hereditary disorder characterized by familial multiple tumors due to mutation in the p53 gene. Affected individuals can develop one or more tumors, including: brain (12%); soft-tissue sarcoma (12%); breast cancer (25%); adrenal tumors (1%); bone cancer (osteosarcoma) (6%); cancer of the lung, prostate, pancreas, and colon as well as lymphoma and melanoma can also occur.

Certain of the most frequently mutated codons are codons 175, 248 and 273, however a variety of oligonucleotides are described below in the atttached table.

Table 11
p53 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
In 2 families with Li-Fraumeni syndrome, there was a C-to-T mutation at the first nucleotide of codon 248 which changed arginine to tryptophan.	GACTGTACCACCATCCACTACAACTACATGTGTAACAGTTCCT GCATGGGCGGCATGAAC**C**GGAGGCCCATCCTCACCATCATC ACACTGGAAGACTCCAGGTCAGGAGCCACTTGCCACC	161
	GGTGGCAAGTGGCTCCTGACCTGGAGTCTTCCAGTGTGATGA TGGTGAGGATGGGCCTCC**G**GTTCATGCCGCCCATGCAGGAA CTGTTACACATGTAGTTGTAGTGGATGGTGGTACAGTC	162
	GCATGAAC**C**GGAGGCCC	163

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GGGCCTCCGGTTCATGC	164
In a family with the Li-Fraumeni syndrome, a G-to-A mutation at the first nucleotide of codon 258 resulting in the substitution of lysine for glutamic acid.	TGTAACAGTTCCTGCATGGGCGGCATGAACCGGAGGCCCAT CCTCACCATCATCACACTGGAAGACTCCAGGTCAGGAGCCAC TTGCCACCCTGCACACTGGCCTGCTGTGCCCCAGCCTC	165
	GAGGCTGGGGCACAGCAGGCCAGTGTGCAGGGTGGCAAGT GGCTCCTGACCTGGAGTCTTCCAGTGTGATGATGGTGAGGAT GGGCCTCCGGTTCATGCCGCCCATGCAGGAACTGTTACA	166
	TCACACTGGAAGACTCC	167
	GGAGTCTTCCAGTGTGA	168
In a family with the Li-Fraumeni syndrome, a G-to-T mutation at the first nucleotide of codon 245 resulting in the substitution of cysteine for glycine.	GTTGGCTCTGACTGTACCACCATCCACTACAACTACATGTGTA ACAGTTCCTGCATGGGCGGCATGAACCGGAGGCCCATCCTC ACCATCATCACACTGGAAGACTCCAGGTCAGGAGCCA	169
A gly245-to-ser, GGC-to-AGC, mutation was found in a patient in whom osteosarcoma was diagnosed at the age of 18 years.	TGGCTCCTGACCTGGAGTCTTCCAGTGTGATGATGGTGAGGA TGGGCCTCCGGTTCATGCCGCCCATGCAGGAACTGTTACACA TGTAGTTGTAGTGGATGGTGGTACAGTCAGAGCCAAC	170
	GCATGGGCGGCATGAAC	171
	GTTCATGCCGCCCATGC	172
In a family with the Li-Fraumeni syndrome, a germline mutation at codon 252: a T-to-C change at the second position resulted in substitution of proline for leucine.	TCCACTACAACTACATGTGTAACAGTTCCTGCATGGGCGGCA TGAACCGGAGGCCCATCCTCACCATCATCACACTGGAAGACT CCAGGTCAGGAGCCACTTGCCACCCTGCACACTGGCC	173
	GGCCAGTGTGCAGGGTGGCAAGTGGCTCCTGACCTGGAGTC TTCCAGTGTGATGATGGTGAGGATGGGCCTCCGGTTCATGCC GCCCATGCAGGAACTGTTACACATGTAGTTGTAGTGGA	174
	GCCCATCCTCACCATCA	175
	TGATGGTGAGGATGGGC	176

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Researchers analyzed for mutations in p53 hepatocellular carcinomas from patients in Qidong, an area of high incidence in China, in which both hepatitis B virus and aflatoxin B1 are risk factors. Eight of 16 tumors had a point mutation at the third base position of codon 249. The G-to-T mutation at codon 249 led to a change from arginine to serine (AGG to AGT).	TACCACCATCCACTACAACTACATGTGTAACAGTTCCTGCATG GGCGGCATGAACCGGAG**G**CCCATCCTCACCATCATCACACT GGAAGACTCCAGGTCAGGAGCCACTTGCCACCCTGCA	177
	TGCAGGGTGGCAAGTGGCTCCTGACCTGGAGTCTTCCAGTG TGATGATGGTGAGGATGGG**C**CTCCGGTTCATGCCGCCCATG CAGGAACTGTTACACATGTAGTTGTAGTGGATGGTGGTA	178
	AACCGGAG**G**CCCATCCT	179
	AGGATGGG**C**CTCCGGTT	180
In cases of hepatocellular carcinoma in southern Africa, a G-to-T substitution in codon 157 resulting in a change from valine to phenylalanine.	CTGGCCAAGACCTGCCCTGTGCAGCTGTGGGTTGATTC**C**ACA CCCCCGCCCGGCACCCGC**G**TCCGCGCCATGGCCATCTACAA GCAGTCACAGCACATGACGGAGGTTGTGAGGCGCTGCC	181
	GGCAGCGCCTCACAACCTCCGTCATGTGCTGTGACTGCTTGT AGATGGCCATGGCGCGGA**C**GCGGGTGCCGGGCGGGGGTGT GGAATCAACCCACAGCTGCACAGGGCAGGTCTTGGCCAG	182
	GCACCCGC**G**TCCGCGCC	183
	GGCGCGGA**C**GCGGGTGC	184
In a family with Li-Fraumeni in which noncancerous skin fibroblasts from affected individuals showed an unusual radiation-resistant phenotype, a point mutation in codon 245 of the P53 gene. A change from GGC to GAC predicted substitution of aspartic acid for glycine.	TTGGCTCTGACTGTACCACCATCCACTACAACTACATGTGTAA CAGTTCCTGCATGGGCG**G**CATGAACCGGAGGCCCATCCTCA CCATCATCACACTGGAAGACTCCAGGTCAGGAGCCAC	185
	GTGGCTCCTGACCTGGAGTCTTCCAGTGTGATGATGGTGAGG ATGGGCCTCCGGTTCATG**C**CGCCCATGCAGGAACTGTTACAC ATGTAGTTGTAGTGGATGGTGGTACAGTCAGAGCCAA	186
	CATGGGCG**G**CATGAACC	187
	GGTTCATG**C**CGCCCATG	188

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
In 2 of 8 families with Li-Fraumeni syndrome, a mutation in codon 248: a CGG-to-CAG change resulting in substitution of glutamine for arginine.	ACTGTACCACCATCCACTACAACTACATGTGTAACAGTTCCTG CATGGGCGGCATGAACCGGAGGCCCATCCTCACCATCATCA CACTGGAAGACTCCAGGTCAGGAGCCACTTGCCACCC	189
	GGGTGGCAAGTGGCTCCTGACCTGGAGTCTTCCAGTGTGAT GATGGTGAGGATGGGCCTCCGGTTCATGCCGCCCATGCAGG AACTGTTACACATGTAGTTGTAGTGGATGGTGGTACAGT	190
	CATGAACCGGAGGCCCA	191
	TGGGCCTCCGGTTCATG	192
In 9 members of an extended family with Li-Fraumeni syndrome, a germline mutation at codon 133 (ATG-to-ACG), resulted in the substitution of threonine for methionine (M133T), and completely cosegregated with the cancer syndrome.	CCCTGACTTTCAACTCTGTCTCCTTCCTCTTCCTACAGTACTC CCCTGCCCTCAACAAGATGTTTTGCCAACTGGCCAAGACCTG CCCTGTGCAGCTGTGGGTTGATTCCACACCCCCGCC	193
	GGCGGGGGTGTGGAATCAACCCACAGCTGCACAGGGCAGGT CTTGGCCAGTTGGCAAAACATCTTGTTGAGGGCAGGGGAGTA CTGTAGGAAGAGGAAGGAGACAGAGTTGAAAGTCAGGG	194
	CAACAAGATGTTTTGCC	195
	GGCAAAACATCTTGTTG	196
In 1 pedigree consistent with the Li-Fraumeni syndrome, a germline G-to-T transversion at codon 272 (valine to leucine) was found.	TCTTGCTTCTCTTTTCCTATCCTGAGTAGTGGTAATCTACTGG GACGGAACAGCTTTGAGGTGCGTGTTTGTGCCTGTCCTGGGA GAGACCGGCGCACAGAGGAAGAGAATCTCCGCAAGA	197
	TCTTGCGGAGATTCTCTTCCTCTGTGCGCCGGTCTCTCCCAG GACAGGCACAAACACGCACCTCAAAGCTGTTCCGTCCCAGTA GATTACCACTACTCAGGATAGGAAAAGAGAAGCAAGA	198
	GCTTTGAGGTGCGTGTT	199
	AACACGCACCTCAAAGC	200
A ser241-to-phe mutation due to a TCC-to-TTC change was found in a patient with hepatoblastoma and multiple foci of osteosarcoma	TTATCTCCTAGGTTGGCTCTGACTGTACCACCATCCACTACAA CTACATGTGTAACAGTTCCTGCATGGGCGGCATGAACCGGAG GCCCATCCTCACCATCATCACACTGGAAGACTCCAG	201
	CTGGAGTCTTCCAGTGTGATGATGGTGAGGATGGGCCTCCG GTTCATGCCGCCCATGCAGGAACTGTTACACATGTAGTTGTA GTGGATGGTGGTACAGTCAGAGCCAACCTAGGAGATAA	202

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAACAGTTCCTGCATGG	203
	CCATGCAGGAACTGTTA	204
An AAG-to-TAG change of codon 120, resulting in conversion from lysine to a stop codon, was found in a patient with osteosarcoma and adenocarcinoma of the lung at age 18 and brain tumor (glioma) at the age of 27.	CAGAAAACCTACCAGGGCAGCTACGGTTTCCGTCTGGGCTTC TTGCATTCTGGGACAGCCAAGTCTGTGACTTGCACGGTCAGT TGCCCTGAGGGGCTGGCTTCCATGAGACTTCAATGCC	205
	GGCATTGAAGTCTCATGGAAGCCAGCCCCTCAGGGCAACTG ACCGTGCAAGTCACAGACTTGGCTGTCCCAGAATGCAAGAAG CCCAGACGGAAACCGTAGCTGCCCTGGTAGGTTTTCTG	206
	GGACAGCCAAGTCTGTG	207
	CACAGACTTGGCTGTCC	208
A CGG-to-TGG change at codon 282, resulting in the substitution of tryptophan for arginine, was found in a patient who developed osteosarcoma at the age of 10 years.	GGTAATCTACTGGGACGGAACAGCTTTGAGGTGCGTGTTTGT GCCTGTCCTGGGAGAGACCGGCGCACAGAGGAAGAGAATCT CCGCAAGAAAGGGGAGCCTCACCACGAGCTGCCCCCAG	209
	CTGGGGGCAGCTCGTGGTGAGGCTCCCCTTTCTTGCGGAGA TTCTCTTCCTCTGTGCGCCGGTCTCTCCCAGGACAGGCACAA ACACGCACCTCAAAGCTGTTCCGTCCCAGTAGATTACC	210
	GGAGAGACCGGCGCACA	211
	TGTGCGCCGGTCTCTCC	212
In 5 of 6 anaplastic carcinomas of the thyroid and in an anaplastic carcinoma thyroid cell line ARO, a CGT-to-CAT mutation converted arginine-273 to histidine.	GCTTCTCTTTTCCTATCCTGAGTAGTGGTAATCTACTGGGACG GAACAGCTTTGAGGTGCGTGTTTGTGCCTGTCCTGGGAGAGA CCGGCGCACAGAGGAAGAGAATCTCCGCAAGAAAGG	213
	CCTTTCTTGCGGAGATTCTCTTCCTCTGTGCGCCGGTCTCTC CCAGGACAGGCACAAACACGCACCTCAAAGCTGTTCCGTCCC AGTAGATTACCACTACTCAGGATAGGAAAAGAGAAGC	214
	TGAGGTGCGTGTTTGTG	215
	CACAAACACGCACCTCA	216

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
A germline GGA-to-GTA mutation resulting in a change of glycine-325 to valine was found in a patient who had non-Hodgkin lymphoma diagnosed at age 17 and colon carcinoma at age 26.	TCCTAGCACTGCCCAACAACACCAGCTCCTCTCCCCAGCCAA AGAAGAAACCACTGGATGGAGAATATTTCACCCTTCAGGTACT AAGTCTTGGGACCTCTTATCAAGTGGAAAGTTTCCA	217
	TGGAAACTTTCCACTTGATAAGAGGTCCCAAGACTTAGTACCT GAAGGGTGAAATATTCTCCATCCAGTGGTTTCTTCTTTGGCTG GGGAGAGGAGCTGGTGTTGTTGGGCAGTGCTAGGA	218
	ACTGGATGGAGAATATT	219
	AATATTCTCCATCCAGT	220
CGC-CCC Arg-72 to Pro association with Lung cancer	AATGGTTCACTGAAGACCCAGGTCCAGATGAAGCTCCCAGAA TGCCAGAGGCTGCTCCCCGCGTGGCCCCTGCACCAGCAGCT CCTACACCGGCGGCCCCTGCACCAGCCCCCTCCTGGCC	221
	GGCCAGGAGGGGGCTGGTGCAGGGGCCGCCGGTGTAGGAG CTGCTGGTGCAGGGGCCACGCGGGGAGCAGCCTCTGGCATT CTGGGAGCTTCATCTGGACCTGGGTCTTCAGTGAACCATT	222
	TGCTCCCCGCGTGGCCC	223
	GGGCCACGCGGGGAGCA	224
CCG-CTG Pro-82 to Leu Breast cancer	AAGCTCCCAGAATGCCAGAGGCTGCTCCCCGCGTGGCCCCT GCACCAGCAGCTCCTACACCGGCGGCCCCTGCACCAGCCCC CTCCTGGCCCCTGTCATCTTCTGTCCCTTCCCAGAAAAC	225
	GTTTTCTGGGAAGGGACAGAAGATGACAGGGGCCAGGAGGG GGCTGGTGCAGGGGCCGCCGGTGTAGGAGCTGCTGGTGCA GGGGCCACGCGGGGAGCAGCCTCTGGCATTCTGGGAGCTT	226
	TCCTACACCGGCGGCCC	227
	GGGCCGCCGGTGTAGGA	228
cCAA-TAA Gln-136 to Term Li-Fraumeni syndrome	TTCAACTCTGTCTCCTTCCTCTTCCTACAGTACTCCCCTGCCC TCAACAAGATGTTTTGCCAACTGGCCAAGACCTGCCCTGTGC AGCTGTGGGTTGATTCCACACCCCCGCCCGGCACCC	229
	GGGTGCCGGGCGGGGGTGTGGAATCAACCCACAGCTGCACA GGGCAGGTCTTGGCCAGTTGGCAAAACATCTTGTTGAGGGCA GGGGAGTACTGTAGGAAGAGGAAGGAGACAGAGTTGAA	230
	TGTTTTGCCAACTGGCC	231
	GGCCAGTTGGCAAAACA	232

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
TGC-TAC Cys-141 to Tyr Li-Fraumeni syndrome	TCCTCTTCCTACAGTACTCCCCTGCCCTCAACAAGATGTTTTG CCAACTGGCCAAGACCT**G**CCCTGTGCAGCTGTGGGTTGATTC **C**ACACCCCCGCCCGGCACCCGCGTCCGCGCCATGGC	233
	GCCATGGCGCGGACGCGGGTGCCGGGCGGGGGTGTGGAAT CAACCCACAGCTGCACAGGG**C**AGGTCTTGGCCAGTTGGCAA AACATCTTGTTGAGGGCAGGGGAGTACTGTAGGAAGAGGA	234
	CAAGACCT**G**CCCTGTGC	235
	GCACAGGG**C**AGGTCTTG	236
aCCC-TCC Pro-151 to Ser Li-Fraumeni syndrome	AACAAGATGTTTTGCCAACTGGCCAAGACCTGCCCTGTGCAG CTGTGGGTTGATTC**C**ACA**C**CCCCGCCCGGCACCCGCGTCCG CGCCATGGCCATCTACAAGCAGTCACAGCACATGACGG	237
	CCGTCATGTGCTGTGACTGCTTGTAGATGGCCATGGCGCGG ACGCGGGTGCCGGGCGGGGG**G**TGTGGAATCAACCCACAGCT GCACAGGGCAGGTCTTGGCCAGTTGGCAAAACATCTTGTT	238
	ATTCCACA**C**CCCCGCCC	239
	GGGCGGGGG**G**TGTGGAAT	240
CCG-CTG Pro-152 to Leu Adrenocortical carcinoma	AGATGTTTTGCCAACTGGCCAAGACCTGCCCTGTGCAGCTGT GGGTTGATTC**C**ACACCCC**C**GCCCGGCACCCGCGTCCGCGCC ATGGCCATCTACAAGCAGTCACAGCACATGACGGAGGT	241
	ACCTCCGTCATGTGCTGTGACTGCTTGTAGATGGCCATGGCG CGGACGCGGGTGCCGGGC**G**GGGGTGTGGAATCAACCCACA GCTGCACAGGGCAGGTCTTGGCCAGTTGGCAAAACATCT	242
	CACACCCC**C**GCCCGGCA	243
	TGCCGGGC**G**GGGGTGTG	244
GGC-GTC Gly-154 to Val Glioblastoma	TTTGCCAACTGGCCAAGACCTGCCCTGTGCAGCTGTGGGTTG ATTC**C**ACACCCCCGCCCG**G**CACCCGCGTCCGCGCCATGGCC ATCTACAAGCAGTCACAGCACATGACGGAGGTTGTGAG	245
	CTCACAACCTCCGTCATGTGCTGTGACTGCTTGTAGATGGCC ATGGCGCGGACGCGGGTG**C**CGGGCGGGGGTGTGGAATCAA CCCACAGCTGCACAGGGCAGGTCTTGGCCAGTTGGCAAA	246
	CCCGCCCG**G**CACCCGCG	247
	CGCGGGTG**C**CGGGCGGG	248
CGC-CAC Arg-175 to His Li-Fraumeni syndrome	CCCGCGTCCGCGCCATGGCCATCTACAAGCAGTCACAGCAC ATGACGGAGGTTGTGAGGC**G**CTGCCCCCACCATGAGCGCTG CTCAGATAGCGATGGTGAGCAGCTGGGGCTGGAGAGACG	249

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CGTCTCTCCAGCCCCAGCTGCTCACCATCGCTATCTGAGCAG CGCTCATGGTGGGGGCAGCGCCTCACAACCTCCGTCATGTG CTGTGACTGCTTGTAGATGGCCATGGCGCGGACGCGGG	250
	TGTGAGGCGCTGCCCCC	251
	GGGGGCAGCGCCTCACA	252
tGAG-AAG Glu-180 to Lys Li-Fraumeni syndrome	ATGGCCATCTACAAGCAGTCACAGCACATGACGGAGGTTGTG AGGCGCTGCCCCCACCATGAGCGCTGCTCAGATAGCGATGG TGAGCAGCTGGGGCTGGAGAGACGACAGGGCTGGTTGC	253
	GCAACCAGCCCTGTCGTCTCTCCAGCCCCAGCTGCTCACCAT CGCTATCTGAGCAGCGCTCATGGTGGGGGCAGCGCCTCACA ACCTCCGTCATGTGCTGTGACTGCTTGTAGATGGCCAT	254
	CCCACCATGAGCGCTGC	255
	GCAGCGCTCATGGTGGG	256
gCGC-TGC Arg-181 to Cys Breast cancer	GCCATCTACAAGCAGTCACAGCACATGACGGAGGTTGTGAGG CGCTGCCCCCACCATGAGCGCTGCTCAGATAGCGATGGTGA GCAGCTGGGGCTGGAGAGACGACAGGGCTGGTTGCCCA	257
	TGGGCAACCAGCCCTGTCGTCTCTCCAGCCCCAGCTGCTCA CCATCGCTATCTGAGCAGCGCTCATGGTGGGGGCAGCGCCT CACAACCTCCGTCATGTGCTGTGACTGCTTGTAGATGGC	258
	ACCATGAGCGCTGCTCA	259
	TGAGCAGCGCTCATGGT	260
CGC-CAC Arg-81 to His Breast cancer	CCATCTACAAGCAGTCACAGCACATGACGGAGGTTGTGAGGC GCTGCCCCCACCATGAGCGCTGCTCAGATAGCGATGGTGAG CAGCTGGGGCTGGAGAGACGACAGGGCTGGTTGCCCAG	261
	CTGGGCAACCAGCCCTGTCGTCTCTCCAGCCCCAGCTGCTC ACCATCGCTATCTGAGCAGCGCTCATGGTGGGGGCAGCGCC TCACAACCTCCGTCATGTGCTGTGACTGCTTGTAGATGG	262
	CCATGAGCGCTGCTCAG	263
	CTGAGCAGCGCTCATGG	264
CAT-CGT His-193 to Arg Li-Fraumeni syndrome	CCAGGGTCCCCAGGCCTCTGATTCCTCACTGATTGCTCTTAG GTCTGGCCCCTCCTCAGCATCTTATCCGAGTGGAAGGAAATT TGCGTGTGGAGTATTTGGATGACAGAAACACTTTTCG	265
	CGAAAAGTGTTTCTGTCATCCAAATACTCCACACGCAAATTTC CTTCCACTCGGATAAGATGCTGAGGAGGGGCCAGACCTAAGA GCAATCAGTGAGGAATCAGAGGCCTGGGGACCCTGG	266

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCCTCAGCATCTTATCC	267
	GGATAAGATGCTGAGGA	268
cCGA-TGA Arg-196 to Term Adrenocortical carcinoma	CCCAGGCCTCTGATTCCTCACTGATTGCTCTTAGGTCTGGCC CCTCCTCAGCATCTTATCCGAGTGGAAGGAAATTTGCGTGTG GAGTATTTGGATGACAGAAACACTTTTCGACATAGTG	269
	CACTATGTCGAAAAGTGTTTCTGTCATCCAAATACTCCACACG CAAATTTCCTTCCACTCGGATAAGATGCTGAGGAGGGGCCAG ACCTAAGAGCAATCAGTGAGGAATCAGAGGCCTGGG	270
	ATCTTATCCGAGTGGAA	271
	TTCCACTCGGATAAGAT	272
cAGA-TGA Arg-209 to Term Li-Fraumeni syndrome	GCCCCTCCTCAGCATCTTATCCGAGTGGAAGGAAATTTGCGT GTGGAGTATTTGGATGACAGAAACACTTTTCGACATAGTGTG GTGGTGCCCTATGAGCCGCCTGAGGTCTGGTTTGCAA	273
	TTGCAAACCAGACCTCAGGCGGCTCATAGGGCACCACCACA CTATGTCGAAAAGTGTTTCTGTCATCCAAATACTCCACACGCA AATTTCCTTCCACTCGGATAAGATGCTGAGGAGGGGC	274
	TGGATGACAGAAACACT	275
	AGTGTTTCTGTCATCCA	276
tCGA-TGA Arg-213 to Term Li-Fraumeni syndrome	CATCTTATCCGAGTGGAAGGAAATTTGCGTGTGGAGTATTTG GATGACAGAAACACTTTTCGACATAGTGTGGTGGTGCCCTAT GAGCCGCCTGAGGTCTGGTTTGCAACTGGGGTCTCTG	277
	CAGAGACCCCAGTTGCAAACCAGACCTCAGGCGGCTCATAG GGCACCACCACACTATGTCGAAAAGTGTTTCTGTCATCCAAAT ACTCCACACGCAAATTTCCTTCCACTCGGATAAGATG	278
	ACACTTTTCGACATAGT	279
	ACTATGTCGAAAAGTGT	280
gCCC-TCC Pro-219 to Ser Adrenocortical carcinoma	GGAAATTTGCGTGTGGAGTATTTGGATGACAGAAACACTTTTC GACATAGTGTGGTGGTGCCCTATGAGCCGCCTGAGGTCTGG TTTGCAACTGGGGTCTCTGGGAGGAGGGGTTAAGGGT	281
	ACCCTTAACCCCTCCTCCCAGAGACCCCAGTTGCAAACCAGA CCTCAGGCGGCTCATAGGGCACCACCACACTATGTCGAAAAG TGTTTCTGTCATCCAAATACTCCACACGCAAATTTCC	282
	TGGTGGTGCCCTATGAG	283
	CTCATAGGGCACCACCA	284

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
TAT-TGT Tyr-220 to Cys Li-Fraumeni syndrome	ATTTGCGTGTGGAGTATTTGGATGACAGAAACACTTTTCGACA TAGTGTGGTGGTGCCCTATGAGCCGCCTGAGGTCTGGTTTG CAACTGGGGTCTCTGGGAGGAGGGGTTAAGGGTGGTT	285
	AACCACCCTTAACCCCTCCTCCCAGAGACCCCAGTTGCAAAC CAGACCTCAGGCGGCTCATAGGGCACCACCACACTATGTCG AAAAGTGTTTCTGTCATCCAAATACTCCACACGCAAAT	286
	GGTGCCCTATGAGCCGC	287
	GCGGCTCATAGGGCACC	288
cTCT-ACT Ser-227 to Thr Rhabdomyosarcoma	CACAGGTCTCCCCAAGGCGCACTGGCCTCATCTTGGGCCTG TGTTATCTCCTAGGTTGGCTCTGACTGTACCACCATCCACTAC AACTACATGTGTAACAGTTCCTGCATGGGCGGCATGA	289
	TCATGCCGCCCATGCAGGAACTGTTACACATGTAGTTGTAGT GGATGGTGGTACAGTCAGAGCCAACCTAGGAGATAACACAG GCCCAAGATGAGGCCAGTGCGCCTTGGGGAGACCTGTG	290
	AGGTTGGCTCTGACTGT	291
	ACAGTCAGAGCCAACCT	292
cCAC-AAC His-233 to Asn Glioma	GCACTGGCCTCATCTTGGGCCTGTGTTATCTCCTAGGTTGGC TCTGACTGTACCACCATCCACTACAACTACATGTGTAACAGTT CCTGCATGGGCGGCATGAACCGGAGGCCCATCCTCA	293
	TGAGGATGGGCCTCCGGTTCATGCCGCCCATGCAGGAACTG TTACACATGTAGTTGTAGTGGATGGTGGTACAGTCAGAGCCA ACCTAGGAGATAACACAGGCCCAAGATGAGGCCAGTGC	294
	CCACCATCCACTACAAC	295
	GTTGTAGTGGATGGTGG	296
cAAC-GAC Asn-235 to Asp Adrenocortical carcinoma	GCCTCATCTTGGGCCTGTGTTATCTCCTAGGTTGGCTCTGAC TGTACCACCATCCACTACAACTACATGTGTAACAGTTCCTGCA TGGGCGGCATGAACCGGAGGCCCATCCTCACCATCA	297
	TGATGGTGAGGATGGGCCTCCGGTTCATGCCGCCCATGCAG GAACTGTTACACATGTAGTTGTAGTGGATGGTGGTACAGTCA GAGCCAACCTAGGAGATAACACAGGCCCAAGATGAGGC	298
	TCCACTACAACTACATG	299
	CATGTAGTTGTAGTGGA	300
AAC-AGC Asn-235 to Ser Rhabdomyosarcoma	CCTCATCTTGGGCCTGTGTTATCTCCTAGGTTGGCTCTGACT GTACCACCATCCACTACAACTACATGTGTAACAGTTCCTGCAT GGGCGGCATGAACCGGAGGCCCATCCTCACCATCAT	301

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATGATGGTGAGGATGGGCCTCCGGTTCATGCCGCCCATGCA GGAACTGTTACACATGTAGTTGTAGTGGATGGTGGTACAGTC AGAGCCAACCTAGGAGATAACACAGGCCCAAGATGAGG	302
	CCACTACAACTACATGT	303
	ACATGTAGTTGTAGTGG	304
ATCc-ATG Ile-251 to Met Glioma	CATCCACTACAACTACATGTGTAACAGTTCCTGCATGGGCGG CATGAACCGGAGGCCCATCCTCACCATCATCACACTGGAAGA CTCCAGGTCAGGAGCCACTTGCCACCCTGCACACTGG	305
	CCAGTGTGCAGGGTGGCAAGTGGCTCCTGACCTGGAGTCTT CCAGTGTGATGATGGTGAGGATGGGCCTCCGGTTCATGCCG CCCATGCAGGAACTGTTACACATGTAGTTGTAGTGGATG	306
	AGGCCCATCCTCACCAT	307
	ATGGTGAGGATGGGCCT	308
ACA-ATA Thr-256 to Ile Glioblastoma	ACATGTGTAACAGTTCCTGCATGGGCGGCATGAACCGGAGG CCCATCCTCACCATCATCACACTGGAAGACTCCAGGTCAGGA GCCACTTGCCACCCTGCACACTGGCCTGCTGTGCCCCA	309
	TGGGGCACAGCAGGCCAGTGTGCAGGGTGGCAAGTGGCTCC TGACCTGGAGTCTTCCAGTGTGATGATGGTGAGGATGGGCCT CCGGTTCATGCCGCCCATGCAGGAACTGTTACACATGT	310
	CATCATCACACTGGAAG	311
	CTTCCAGTGTGATGATG	312
CTG-CAG Leu-257 to Gln Li-Fraumeni syndrome	TGTGTAACAGTTCCTGCATGGGCGGCATGAACCGGAGGCCC ATCCTCACCATCATCACACTGGAAGACTCCAGGTCAGGAGCC ACTTGCCACCCTGCACACTGGCCTGCTGTGCCCCAGCC	313
	GGCTGGGGCACAGCAGGCCAGTGTGCAGGGTGGCAAGTGG CTCCTGACCTGGAGTCTTCCAGTGTGATGATGGTGAGGATGG GCCTCCGGTTCATGCCGCCCATGCAGGAACTGTTACACA	314
	CATCACACTGGAAGACT	315
	AGTCTTCCAGTGTGATG	316
CTG-CCG Leu-265 to Pro Li-Fraumeni syndrome	GACCTGATTTCCTTACTGCCTCTTGCTTCTCTTTTCCTATCCT GAGTAGTGGTAATCTACTGGGACGGAACAGCTTTGAGGTGCG TGTTTGTGCCTGTCCTGGGAGAGACCGGCGCACAGA	317
	TCTGTGCGCCGGTCTCTCCCAGGACAGGCACAAACACGCAC CTCAAAGCTGTTCCGTCCCAGTAGATTACCACTACTCAGGAT AGGAAAAGAGAAGCAAGAGGCAGTAAGGAAATCAGGTC	318

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAATCTACTGGGACGGA	319
	TCCGTCCCAGTAGATTA	320
gCGT-TGT Arg-273 to Cys Li-Fraumeni syndrome	TGCTTCTCTTTTCCTATCCTGAGTAGTGGTAATCTACTGGGAC GGAACAGCTTTGAGGTGCGTGTTTGTGCCTGTCCTGGGAGA GACCGGCGCACAGAGGAAGAGAATCTCCGCAAGAAAG	321
	CTTTCTTGCGGAGATTCTCTTCCTCTGTGCGCCGGTCTCTCC CAGGACAGGCACAAACACGCACCTCAAAGCTGTTCCGTCCCA GTAGATTACCACTACTCAGGATAGGAAAAGAGAAGCA	322
	TTGAGGTGCGTGTTTGT	323
	ACAAACACGCACCTCAA	324
TGT-TAT Cys-275 to Tyr Li-Fraumeni syndrome	CTTTTCCTATCCTGAGTAGTGGTAATCTACTGGGACGGAACA GCTTTGAGGTGCGTGTTTGTGCCTGTCCTGGGAGAGACCGG CGCACAGAGGAAGAGAATCTCCGCAAGAAAGGGGAGCC	325
	GGCTCCCCTTTCTTGCGGAGATTCTCTTCCTCTGTGCGCCGG TCTCTCCCAGGACAGGCACAAACACGCACCTCAAAGCTGTTC CGTCCCAGTAGATTACCACTACTCAGGATAGGAAAAG	326
	GCGTGTTTGTGCCTGTC	327
	GACAGGCACAAACACGC	328
CCT-CTT Pro-278 to Leu Breast cancer	TCCTGAGTAGTGGTAATCTACTGGGACGGAACAGCTTTGAGG TGCGTGTTTGTGCCTGTCCTGGGAGAGACCGGCGCACAGAG GAAGAGAATCTCCGCAAGAAAGGGGAGCCTCACCACGA	329
	TCGTGGTGAGGCTCCCCTTTCTTGCGGAGATTCTCTTCCTCT GTGCGCCGGTCTCTCCCAGGACAGGCACAAACACGCACCTC AAAGCTGTTCCGTCCCAGTAGATTACCACTACTCAGGA	330
	TGCCTGTCCTGGGAGAG	331
	CTCTCCCAGGACAGGCA	332
AGA-AAA Arg-280 to Lys Glioma	GTAGTGGTAATCTACTGGGACGGAACAGCTTTGAGGTGCGTG TTTGTGCCTGTCCTGGGAGAGACCGGCGCACAGAGGAAGAG AATCTCCGCAAGAAAGGGGAGCCTCACCACGAGCTGCC	333
	GGCAGCTCGTGGTGAGGCTCCCCTTTCTTGCGGAGATTCTCT TCCTCTGTGCGCCGGTCTCTCCCAGGACAGGCACAAACACG CACCTCAAAGCTGTTCCGTCCCAGTAGATTACCACTAC	334
	TCCTGGGAGAGACCGGC	335
	GCCGGTCTCTCCCAGGA	336

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
GAA-GCA Glu-286 to Ala Adrenocortical carcinoma	GGAACAGCTTTGAGGTGCGTGTTTGTGCCTGTCCTGGGAGA GACCGGCGCACAGAGGAAGAGAATCTCCGCAAGAAAGGGGA GCCTCACCACGAGCTGCCCCCAGGGAGCACTAAGCGAGG	337
	CCTCGCTTAGTGCTCCCTGGGGGCAGCTCGTGGTGAGGCTC CCCTTTCTTGCGGAGATTCTCTTCCTCTGTGCGCCGGTCTCT CCCAGGACAGGCACAAACACGCACCTCAAAGCTGTTCC	338
	AGAGGAAGAGAATCTCC	339
	GGAGATTCTCTTCCTCT	340
CGA-CCA Arg-306 to Pro Rhabdomyosarcoma	AAGAGAATCTCCGCAAGAAAGGGGAGCCTCACCACGAGCTG CCCCCAGGGAGCACTAAGCGAGGTAAGCAAGCAGGACAAGA AGCGGTGGAGGAGACCAAGGGTGCAGTTATGCCTCAGAT	341
	ATCTGAGGCATAACTGCACCCTTGGTCTCCTCCACCGCTTCT TGTCCTGCTTGCTTACCTCGCTTAGTGCTCCCTGGGGGCAGC TCGTGGTGAGGCTCCCCTTTCTTGCGGAGATTCTCTT	342
	CACTAAGCGAGGTAAGC	343
	GCTTACCTCGCTTAGTG	344
gCGA-TGA Arg-306 to Term Li-Fraumeni syndrome	GAAGAGAATCTCCGCAAGAAAGGGGAGCCTCACCACGAGCT GCCCCCAGGGAGCACTAAGCGAGGTAAGCAAGCAGGACAAG AAGCGGTGGAGGAGACCAAGGGTGCAGTTATGCCTCAGA	345
	TCTGAGGCATAACTGCACCCTTGGTCTCCTCCACCGCTTCTT GTCCTGCTTGCTTACCTCGCTTAGTGCTCCCTGGGGGCAGCT CGTGGTGAGGCTCCCCTTTCTTGCGGAGATTCTCTTC	346
	GCACTAAGCGAGGTAAG	347
	CTTACCTCGCTTAGTGC	348
gCGC-TGC Arg-337 to Cys Osteosarcoma	GGTACTGTGAATATACTTACTTCTCCCCCTCCTCTGTTGCTGC AGATCCGTGGGCGTGAGCGCTTCGAGATGTTCCGAGAGCTG AATGAGGCCTTGGAACTCAAGGATGCCCAGGCTGGGA	349
	TCCCAGCCTGGGCATCCTTGAGTTCCAAGGCCTCATTCAGCT CTCGGAACATCTCGAAGCGCTCACGCCCACGGATCTGCAGC AACAGAGGAGGGGGAGAAGTAAGTATATTCACAGTACC	350
	GGCGTGAGCGCTTCGAG	351
	CTCGAAGCGCTCACGCC	352
CTG-CCG Leu-344 to Pro Li-Fraumeni syndrome	CTCCCCCTCCTCTGTTGCTGCAGATCCGTGGGCGTGAGCGC TTCGAGATGTTCCGAGAGCTGAATGAGGCCTTGGAACTCAAG GATGCCCAGGCTGGGAAGGAGCCAGGGGGGAGCAGGGC	353

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCCCTGCTCCCCCCTGGCTCCTTCCCAGCCTGGGCATCCTT GAGTTCCAAGGCCTCATTCAGCTCTCGGAACATCTCGAAGCG CTCACGCCCACGGATCTGCAGCAACAGAGGAGGGGGAG	354
	CCGAGAGCTGAATGAGG	355
	CCTCATTCAGCTCTCGG	356

EXAMPLE 6
beta globin

Hemoglobin, the major protein in the red blood cell, binds oxygen reversibly and is responsible for the cells' capacity to transport oxygen to the tissues. In adults, the major hemoglobin is hemoglobin A, a tetrameric protein consisting of two identical alpha globin chains and two beta globin chains. Disorders involving hemoglobin are among the most common genetic disorders worldwide, with approximately 5% of the world's population being carriers for clinically important hemoglobin mutations. Approximately 300,000 severely affected homozygotes or compound heterozygotes are born each year.

Mutation of the glutamic acid at position 7 in beta globin to valine causes sickle cell anemia, the clinical manifestations of which are well known. Mutations that cause absence of beta chain cause beta-zero-thalassemia. Reduced amounts of detectable beta globin causes beta-plus-thalassemia. For clinical purposes, beta-thalassemia is divided into thalassemia major (transfusion dependent), thalassemia intermedia (of intermediate severity), and thalassemia minor (asymptomatic). Patients with thalassemia major present in the first year of life with severe anemia; they are unable to maintain a hemoglobin level about 5 gm/dl.

The beta-thalassemias were among the first human genetic diseases to be examined by means of recombinant DNA analysis. Baysal et al., *Hemoglobin* 19(3-4):213-36 (1995) and others provide a compendium of mutations that result in beta-thalassemia.

Hemoglobin disorders were among the first to be considered for gene therapy. Transcriptional silencing of genes transferred into hematopoietic stem cells, however, poses one of the most significant challenges to its success. If the transferred gene is not completely silenced, a progressive decline in gene expression is often observed. Position effect variegation (PEV) and silencing mechanisms may act on a transferred globin gene residing in chromatin outside of the normal globin locus during the important terminal phases of erythroblast development when globin transcripts normally

accumulate rapidly despite heterochromatization and shutdown of the rest of the genome. The attached table discloses the correcting oligonucleotide base sequences for the beta globin oligonucleotides of the invention.

Table 12
Beta Globin Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Sickle Cell Anemia GLU-7-VAL GAG to GTG	TCTGACACAACTGTGTTCACTAGCAACCTCAAACAGACACCA TGGTGCACCTGACTCCTGAGGAGAAGTCTGCCGTTACTGCC CTGTGGGGCAAGGTGAACGTGGATGAAGTTGGTGGTGA	357
	TCACCACCAACTTCATCCACGTTCACCTTGCCCCACAGGGCA GTAACGGCAGACTTCTCCTCAGGAGTCAGGTGCACCATGGT GTCTGTTTGAGGTTGCTAGTGAACACAGTTGTGTCAGA	358
	GACTCCTGAGGAGAAGT	359
	ACTTCTCCTCAGGAGTC	360
Thalassaemia Beta MET-0-ARG ATG to AGG	CTATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGCA ACCTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGA AGTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGT	361
	ACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCTC CTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGCT AGTGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATAG	362
	AGACACCATGGTGCACC	363
	GGTGCACCATGGTGTCT	364
Thalassaemia Beta MET-0-ILE ATG to ATA	TATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGCAA CCTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGAA GTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGTG	365
	CACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCT CCTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGC TAGTGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATA	366
	GACACCATGGTGCACCT	367
	AGGTGCACCATGGTGTC	368
Thalassaemia Beta MET-0-ILE ATG to ATT	TATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGCAA CCTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAC[illegible]A GTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGTG	369

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCT CCTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGC TAGTGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATA	370
	GACACCATGGTGCACCT	371
	AGGTGCACCATGGTGTC	372
Thalassaemia Beta MET-0-LYS ATG to AAG	CTATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGCA ACCTCAAACAGACACCATTGGTGCACCTGACTCCTGAGGAGA AGTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGT	373
	ACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCTC CTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGCT AGTGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATAG	374
	AGACACCATTGGTGCACC	375
	GGTGCACCATGGTGTCT	376
Thalassaemia Beta MET-0-THR ATG to ACG	CTATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGCA ACCTCAAACAGACACCATTGGTGCACCTGACTCCTGAGGAGA AGTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGT	377
	ACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCTC CTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGCT AGTGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATAG	378
	AGACACCATTGGTGCACC	379
	GGTGCACCATGGTGTCT	380
Thalassaemia Beta MET-0-VAL ATG to GTG	TCTATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGC AACCTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAG AAGTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACG	381
	CGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCTCC TCAGGAGTCAGGTGCACCATTGGTGTCTGTTTGAGGTTGCTAG TGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATAGA	382
	CAGACACCATGGTGCAC	383
	GTGCACCATTGGTGTCTG	384
Thalassaemia Beta TRP-16-Term TGG to TGA	TCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGAAGT CTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGTGGATGAA GTTGGTGGTGAGGCCCTGGGCAGGTTGGTATCAAGGTTA	385
	TAACCTTGATACCAACCTGCCCAGGGCCTCACCACCAACTTC ATCCACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACT TCTCCTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGA	386

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCCCTGTGGGGCAAGGT	387
	ACCTTGCCCCACAGGGC	388
Thalassaemia Beta TRP-16-Term TGG to TAG	CTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGAAG TCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGTGGATGA AGTTGGTGGTGAGGCCCTGGGCAGGTTGGTATCAAGGTT	389
	AACCTTGATACCAACCTGCCCAGGGCCTCACCACCAACTTCA TCCACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTT CTCCTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAG	390
	TGCCCTGTGGGGCAAGG	391
	CCTTGCCCCACAGGGCA	392
Thalassaemia Beta LYS-18-Term AAG to TAG	ACAGACACCATGGTGCACCTGACTCCTGAGGAGAAGTCTGC CGTTACTGCCCTGTGGGGCAAGGTGAACGTGGATGAAGTTG GTGGTGAGGCCCTGGGCAGGTTGGTATCAAGGTTACAAG	393
	CTTGTAACCTTGATACCAACCTGCCCAGGGCCTCACCACCAA CTTCATCCACGTTCACCTTGCCCCACAGGGCAGTAACGGCA GACTTCTCCTCAGGAGTCAGGTGCACCATGGTGTCTGT	394
	TGTGGGGCAAGGTGAAC	395
	GTTCACCTTGCCCCACA	396
Thalassaemia Beta ASN-20-SER AAC to AGC	CCATGGTGCACCTGACTCCTGAGGAGAAGTCTGCCGTTACT GCCCTGTGGGGCAAGGTGAACGTGGATGAAGTTGGTGGTGA GGCCCTGGGCAGGTTGGTATCAAGGTTACAAGACAGGTT	397
	AACCTGTCTTGTAACCTTGATACCAACCTGCCCAGGGCCTCA CCACCAACTTCATCCACGTTCACCTTGCCCCACAGGGCAGTA ACGGCAGACTTCTCCTCAGGAGTCAGGTGCACCATGG	398
	CAAGGTGAACGTGGATG	399
	CATCCACGTTCACCTTG	400
Thalassaemia Beta GLU-23-ALA GAA to GCA	ACCTGACTCCTGAGGAGAAGTCTGCCGTTACTGCCCTGTGG GGCAAGGTGAACGTGGATGAAGTTGGTGGTGAGGCCCTGG GCAGGTTGGTATCAAGGTTACAAGACAGGTTTAAGGAGAC	401
	GTCTCCTTAAACCTGTCTTGTAACCTTGATACCAACCTGCCC AGGGCCTCACCACCAACTTCATCCACGTTCACCTTGCCCCAC AGGGCAGTAACGGCAGACTTCTCCTCAGGAGTCAGGT	402
	CGTGGATGAAGTTGGTG	403
	CACCAACTTCATCCACG	404

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia Beta GLU-23-term GAA to TAA	CACCTGACTCCTGAGGAGAAGTCTGCCGTTACTGCCCTGTG GGGCAAGGTGAACGTGGATGAAGTTGGTGGTGAGGCCCTG GGCAGGTTGGTATCAAGGTTACAAGACAGGTTTAAGGAGA	405
	TCTCCTTAAACCTGTCTTGTAACCTTGATACCAACCTGCCCA GGGCCTCACCACCAACTTCATCCACGTTCACCTTGCCCCACA GGGCAGTAACGGCAGACTTCTCCTCAGGAGTCAGGTG	406
	ACGTGGATGAAGTTGGT	407
	ACCAACTTCATCCACGT	408
Thalassaemia Beta GLU-27-LYS GAG to AAG	GAGGAGAAGACTGCTGTCAATGCCCTGTGGGGCAAAGTGAA CGTGGATGCAGTTGGTGGTGAGGCCCTGGGCAGGTTGGTAT CAAGGTTATAAGAGAGGCTCAAGGAGGCAAATGGAAACT	409
	AGTTTCCATTTGCCTCCTTGAGCCTCTCTTATAACCTTGATAC CAACCTGCCCAGGGCCTCACCACCAACTGCATCCACGTTCA CTTTGCCCCACAGGGCATTGACAGCAGTCTTCTCCTC	410
	TTGGTGGTGAGGCCCTG	411
	CAGGGCCTCACCACCAA	412
Thalassaemia Beta GLU-27-Term GAG to TAG	GAGGAGAAGACTGCTGTCAATGCCCTGTGGGGCAAAGTGAA CGTGGATGCAGTTGGTGGTGAGGCCCTGGGCAGGTTGGTAT CAAGGTTATAAGAGAGGCTCAAGGAGGCAAATGGAAACT	413
	AGTTTCCATTTGCCTCCTTGAGCCTCTCTTATAACCTTGATAC CAACCTGCCCAGGGCCTCACCACCAACTGCATCCACGTTCA CTTTGCCCCACAGGGCATTGACAGCAGTCTTCTCCTC	414
	TTGGTGGTGAGGCCCTG	415
	CAGGGCCTCACCACCAA	416
Thalassaemia Beta ALA-28-SER GCC to TCC	GAGAAGACTGCTGTCAATGCCCTGTGGGGCAAAGTGAACGT GGATGCAGTTGGTGGTGAGGCCCTGGGCAGGTTGGTATCAA GGTTATAAGAGAGGCTCAAGGAGGCAAATGGAAACTGGG	417
	CCCAGTTTCCATTTGCCTCCTTGAGCCTCTCTTATAACCTTGA TACCAACCTGCCCAGGGCCTCACCACCAACTGCATCCACGT TCACTTTGCCCCACAGGGCATTGACAGCAGTCTTCTC	418
	GTGGTGAGGCCCTGGGC	419
	GCCCAGGGCCTCACCAC	420

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia Beta ARG-31-THR AGG to ACG	CTGTCAATGCCCTGTGGGGCAAAGTGAACGTGGATGCAGTT GGTGGTGAGGCCCTGGGCA**G**GTTGGTATCAAGGTTATAAGA GAGGCTCAAGGAGGCAAATGGAAACTGGGCATGTGTAGA	421
	TCTACACATGCCCAGTTTCCATTTGCCTCCTTGAGCCTCTCTT ATAACCTTGATACCAAC**C**TGCCCAGGGCCTCACCACCAACTG CATCCACGTTCACTTTGCCCCACAGGGCATTGACAG	422
	CCTGGGCA**G**GTTGGTAT	423
	ATACCAAC**C**TGCCCAGG	424
Thalassaemia Beta Leu-33-GLN CTG to CAG	TGGGTTTCTGATAGGCACTGACTCTCTGTCCCTTGGGCTGTT TTCCTACCCTCAGATTAC**T**GGTGGTCTACCCTTGGACCCAGA GGTTCTTTGAGTCCTTTGGGGATCTGTCCTCTCCTGA	425
	TCAGGAGAGGACAGATCCCCAAAGGACTCAAAGAACCTCTG GGTCCAAGGGTAGACCACC**A**GTAATCTGAGGGTAGGAAAAC AGCCCAAGGGACAGAGAGTCAGTGCCTATCAGAAACCCA	426
	CAGATTAC**T**GGTGGTCT	427
	AGACCACC**A**GTAATCTG	428
Thalassaemia Beta TYR-36-Term TAC to TAA	ATAGGCACTGACTCTCTGTCCCTTGGGCTGTTTTCCTACCCT CAGATTACTGGTGGTCTA**C**CCTTGGACCCAGAGGTTCTTTGA GTCCTTTGGGGATCTGTCCTCTCCTGATGCTGTTATG	429
	CATAACAGCATCAGGAGAGGACAGATCCCCAAAGGACTCAAA GAACCTCTGGGTCCAAGG**G**TAGACCACCAGTAATCTGAGGG TAGGAAAACAGCCCAAGGGACAGAGAGTCAGTGCCTAT	430
	GTGGTCTA**C**CCTTGGAC	431
	GTCCAAGG**G**TAGACCAC	432
Thalassaemia Beta TRP-38-Term TGG to TGA	ACTGACTCTCTGTCCCTTGGGCTGTTTTCCTACCCTCAGATT ACTGGTGGTCTACCCTTG**G**ACCCAGAGGTTCTTTGAGTCCTT TGGGGATCTGTCCTCTCCTGATGCTGTTATGGGCAAC	433
	GTTGCCCATAACAGCATCAGGAGAGGACAGATCCCCAAAGG ACTCAAAGAACCTCTGGGT**C**CAAGGGTAGACCACCAGTAATC TGAGGGTAGGAAAACAGCCCAAGGGACAGAGAGTCAGT	434
	TACCCTTG**G**ACCCAGAG	435
	CTCTGGGT**C**CAAGGGTA	436
Thalassaemia Beta TRP-38-Term TGG to TAG	CACTGACTCTCTGTCCCTTGGGCTGTTTTCCTACCCTCAGAT TACTGGTGGTCTACCCTT**G**GACCCAGAGGTTCTTTGAGTCCT TTGGGGATCTGTCCTCTCCTGATGCTGTTATGGGCAA	437

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTGCCCATAACAGCATCAGGAGAGGACAGATCCCCAAAGGA CTCAAAGAACCTCTGGGTCCAAGGGTAGACCACCAGTAATCT GAGGGTAGGAAAACAGCCCAAGGGACAGAGAGTCAGTG	438
	CTACCCTTGGACCCAGA	439
	TCTGGGTCCAAGGGTAG	440
Thalassaemia Beta GLN-40-Term CAG-TAG	ACTCTCTGTCCCTTGGGCTGTTTTCCTACCCTCAGATTACTG GTGGTCTACCCTTGGACCCAGAGGTTCTTTGAGTCCTTTGGG GATCTGTCCTCTCCTGATGCTGTTATGGGCAACCCTA	441
	TAGGGTTGCCCATAACAGCATCAGGAGAGGACAGATCCCCA AAGGACTCAAAGAACCTCTGGGTCCAAGGGTAGACCACCAG TAATCTGAGGGTAGGAAAACAGCCCAAGGGACAGAGAGT	442
	CTTGGACCCAGAGGTTC	443
	GAACCTCTGGGTCCAAG	444
Thalassaemia Beta GLU-44-Term GAG to TAG	TTGGGCTGTTTTCCTACCCTCAGATTACTGGTGGTCTACCCT TGGACCCAGAGGTTCTTTGAGTCCTTTGGGGATCTGTCCTCT CCTGATGCTGTTATGGGCAACCCTAAGGTGAAGGCTC	445
	GAGCCTTCACCTTAGGGTTGCCCATAACAGCATCAGGAGAG GACAGATCCCCAAAGGACTCAAAGAACCTCTGGGTCCAAGG GTAGACCACCAGTAATCTGAGGGTAGGAAAACAGCCCAA	446
	GGTTCTTTGAGTCCTTT	447
	AAAGGACTCAAAGAACC	448
Thalassaemia Beta LYS-62-Term AAG to TAG	TTCTTTGAGTCCTTTGGGGATCTGTCCTCTCCTGATGCTGTTA TGGGCAACCCTAAGGTGAAGGCTCATGGCAAGAAGGTGCTA GGTGCCTTTAGTGATGGCCTGGCTCACCTGGACAACC	449
	GGTTGTCCAGGTGAGCCAGGCCATCACTAAAGGCACCTAGC ACCTTCTTGCCATGAGCCTTCACCTTAGGGTTGCCCATAACA GCATCAGGAGAGGACAGATCCCCAAAGGACTCAAAGAA	450
	CTAAGGTGAAGGCTCAT	451
	ATGAGCCTTCACCTTAG	452
Thalassaemia Beta SER-73-ARG AGT to AGA	TGCTGTTATGGGCAACCCTAAGGTGAAGGCTCATGGCAAGA AGGTGCTAGGTGCCTTTAGTGATGGCCTGGCTCACCTGGAC AACCTCAAGGGCACTTTTTCTCAGCTGAGTGAGCTGCAC	453
	GTGCAGCTCACTCAGCTGAGAAAAAGTGCCCTTGAGGTTGTC CAGGTGAGCCAGGCCATCACTAAAGGCACCTAGCACCTTCT TGCCATGAGCCTTCACCTTAGGGTTGCCCATAACAGCA	454

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCCTTTAGTGATGGCCT	455
	AGGCCATCACTAAAGGC	456
Haemolytic Anaemia GLY-75-VAL GGC to GTC	TTATGGGCAACCCTAAGGTGAAGGCTCATGGCAAGAAGGTG CTAGGTGCCTTTAGTGATGGCCTGGCTCACCTGGACAACCT CAAGGGCACTTTTTCTCAGCTGAGTGAGCTGCACTGTGA	457
	TCACAGTGCAGCTCACTCAGCTGAGAAAAAGTGCCCTTGAG GTTGTCCAGGTGAGCCAGGCCATCACTAAAGGCACCTAGCA CCTTCTTGCCATGAGCCTTCACCTTAGGGTTGCCCATAA	458
	TAGTGATGGCCTGGCTC	459
	GAGCCAGGCCATCACTA	460
Thalassaemia Beta GLU-91-Term GAG to TAG	GCCTTTAGTGATGGCCTGGCTCACCTGGACAACCTCAAGGG CACCTTTGCCACACTGAGTGAGCTGCACTGTGACAAGCTGC ACGTGGATCCTGAGAACTTCAGGGTGAGTCTATGGGACC	461
	GGTCCCATAGACTCACCCTGAAGTTCTCAGGATCCACGTGCA GCTTGTCACAGTGCAGCTCACTCAGTGTGGCAAAGGTGCCC TTGAGGTTGTCCAGGTGAGCCAGGCCATCACTAAAGGC	462
	CACTGAGTGAGCTGCAC	463
	GTGCAGCTCACTCAGTG	464
Thalassaemia Beta VAL-99-MET GTG to ATG	CTGGACAACCTCAAGGGCACTTTTTCTCAGCTGAGTGAGCTG CACTGTGACAAGCTGCACGTGGATCCTGAGAACTTCAGGGT GAGTCCAGGAGATGCTTCACTTTTCTCTTTTTACTTTC	465
	GAAAGTAAAAAGAGAAAAGTGAAGCATCTCCTGGACTCACCC TGAAGTTCTCAGGATCCACGTGCAGCTTGTCACAGTGCAGCT CACTCAGCTGAGAAAAAGTGCCCTTGAGGTTGTCCAG	466
	AGCTGCACGTGGATCCT	467
	AGGATCCACGTGCAGCT	468
Thalassaemia Beta LEU-111-PRO CTG-CCG	CCCTTTTGCTAATCATGTTCATACCTCTTATCTTCCTCCCACA GCTCCTGGGCAACGTGCTGGTCTGTGTGCTGGCCCATCACT TTGGCAAAGAATTCACCCCACCAGTGCAGGCTGCCTA	469
	TAGGCAGCCTGCACTGGTGGGGTGAATTCTTTGCCAAAGTG ATGGGCCAGCACACAGACCAGCACGTTGCCCAGGAGCTGTG GGAGGAAGATAAGAGGTATGAACATGATTAGCAAAAGGG	470
	CAACGTGCTGGTCTGTG	471
	CACAGACCAGCACGTTG	472

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia Beta CYS-113-Term TGT to TGA	GCTAATCATGTTCATACCTCTTATCTTCCTCCCACAGCTCCTG GGCAACGTGCTGGTCTGTGTGCTGGCCCATCACTTTGGCAA AGAATTCACCCCACCAGTGCAGGCTGCCTATCAGAAA	473
	TTTCTGATAGGCAGCCTGCACTGGTGGGGTGAATTCTTTGCC AAAGTGATGGGCCAGCACACAGACCAGCACGTTGCCCAGGA GCTGTGGGAGGAAGATAAGAGGTATGAACATGATTAGC	474
	CTGGTCTGTGTGCTGGC	475
	GCCAGCACACAGACCAG	476
Thalassaemia Beta LEU-115-PRO CTG to CCG	TCATGTTCATACCTCTTATCTTCCTCCCACAGCTCCTGGGCA ACGTGCTGGTCTGTGTGCTGGCCCATCACTTTGGCAAAGAAT TCACCCCACCAGTGCAGGCTGCCTATCAGAAAGTGGT	477
	ACCACTTTCTGATAGGCAGCCTGCACTGGTGGGGTGAATTCT TTGCCAAAGTGATGGGCCAGCACACAGACCAGCACGTTGCC CAGGAGCTGTGGGAGGAAGATAAGAGGTATGAACATGA	478
	CTGTGTGCTGGCCCATC	479
	GATGGGCCAGCACACAG	480
Thalassaemia Beta ALA-116-ASP GCC to GAC	TGTTCATACCTCTTATCTTCCTCCCACAGCTCCTGGGCAACG TGCTGGTCTGTGTGCTGGCCCATCACTTTGGCAAAGAATTCA CCCCACCAGTGCAGGCTGCCTATCAGAAAGTGGTGGC	481
	GCCACCACTTTCTGATAGGCAGCCTGCACTGGTGGGGTGAA TTCTTTGCCAAAGTGATGGGCCAGCACACAGACCAGCACGTT GCCCAGGAGCTGTGGGAGGAAGATAAGAGGTATGAACA	482
	TGTGCTGGCCCATCACT	483
	AGTGATGGGCCAGCACA	484
Thalassaemia Beta GLU-122-Term GAA to TAA	TTCCTCCCACAGCTCCTGGGCAACGTGCTGGTCTGTGTGCT GGCCCATCACTTTGGCAAAGAATTCACCCCACCAGTGCAGG CTGCCTATCAGAAAGTGGTGGCTGGTGTGGCTAATGCCC	485
	GGGCATTAGCCACACCAGCCACCACTTTCTGATAGGCAGCC TGCACTGGTGGGGTGAATTCTTTGCCAAAGTGATGGGCCAG CACACAGACCAGCACGTTGCCCAGGAGCTGTGGGAGGAA	486
	TTGGCAAAGAATTCACC	487
	GGTGAATTCTTTGCCAA	488
Thalassaemia Beta GLN-128-PRO CAG to CCG	GCAACGTGCTGGTCTGTGTGCTGGCCCATCACTTTGGCAAA GAATTCACCCCACCAGTGCAGGCTGCCTATCAGAAAGTGGT GGCTGGTGTGGCTAATGCCCTGGCCCACAAGTATCACTA	489

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAGTGATACTTGTGGGCCAGGGCATTAGCCACACCAGCCAC CACTTTCTGATAGGCAGCCTGCACTGGTGGGGTGAATTCTTT GCCAAAGTGATGGGCCAGCACACAGACCAGCACGTTGC	490
	ACCAGTGCAGGCTGCCT	491
	AGGCAGCCTGCACTGGT	492
Thalassaemia Beta GLN-128-Term CAG to TAG	GGCAACGTGCTGGTCTGTGTGCTGGCCCATCACTTTGGCAA AGAATTCACCCCACCAGTGCAGGCTGCCTATCAGAAAGTGGT GGCTGGTGTGGCTAATGCCCTGGCCCACAAGTATCACT	493
	AGTGATACTTGTGGGCCAGGGCATTAGCCACACCAGCCACC ACTTTCTGATAGGCAGCCTGCACTGGTGGGGTGAATTCTTTG CCAAAGTGATGGGCCAGCACACAGACCAGCACGTTGCC	494
	CACCAGTGCAGGCTGCC	495
	GGCAGCCTGCACTGGTG	496
Thalassaemia Beta GLN-132-LYS CAG to AAG	GTCTGTGTGCTGGCCCATCACTTTGGCAAAGAATTCACCCCA CCAGTGCAGGCTGCCTATCAGAAAGTGGTGGCTGGTGTGGC TAATGCCCTGGCCCACAAGTATCACTAAGCTCGCTTTC	497
	GAAAGCGAGCTTAGTGATACTTGTGGGCCAGGGCATTAGCC ACACCAGCCACCACTTTCTGATAGGCAGCCTGCACTGGTGG GGTGAATTCTTTGCCAAAGTGATGGGCCAGCACACAGAC	498
	CTGCCTATCAGAAAGTG	499
	CACTTTCTGATAGGCAG	500

EXAMPLE 7
Retinoblastoma

Retinoblastoma (RB) is an embryonic neoplasm of retinal origin. It almost always presents in early childhood and is often bilateral. The risk of osteogenic sarcoma is increased 500-fold in bilateral retinoblastoma patients, the bone malignancy being at sites removed from those exposed to radiation treatment of the eye tumor.

The retinoblastoma susceptibility gene (pRB; pRb) plays a pivotal role in the regulation of the cell cycle. pRB restrains cell cycle progression by maintaining a checkpoint in late G_1 that controls commitment of cells to enter S phase. The critical role that pRB plays in cell cycle regulation explains its

status as archetypal tumor suppressor: loss of pRB function results in an inability to maintain control of the G_1 checkpoint; unchecked progression through the cell cycle is, in turn, a hallmark of neoplasia.

Blanquet *et al., Hum. Molec. Genet.* 4: 383-388 (1995) performed a mutation survey of the RB1 gene in 232 patients with hereditary or nonhereditary retinoblastoma. They systematically explored all 27 exons and flanking sequences, as well as the promoter. All types of point mutations were represented and found to be unequally distributed along the RB1 gene sequence. In the population studied, exons 3, 8, 18, and 19 were preferentially altered. The attached table discloses the correcting oligonucleotide base sequences for the retinoblastoma oligonucleotides of the invention.

Table 13
pRB Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Trp99Term TGG-TAG	AATATTTGATCTTTATTTTTTGTTCCCAGGGAGGTTATATTCAA AAGAAAAAGGAACTGT**G**GGGAATCTGTATCTTTATTGCAGCA GTTGACCTAGATGAGATGTCGTTCACTTTTACTGA	501
	TCAGTAAAAGTGAACGACATCTCATCTAGGTCAACTGCTGCA ATAAAGATACAGATTCCC**C**ACAGTTCCTTTTTCTTTTGAATATA ACCTCCCTGGGAACAAAAAATAAAGATCAAATATT	502
	GGAACTGT**G**GGGAATCT	503
	AGATTCCC**C**ACAGTTCC	504
Retinoblastoma Glu137Asp GAA-GAT	ATTTACTTTTTTCTATTCTTTCCTTTGTAGTGTCCATAAATTCTT TAACTTACTAAAAGA**A**ATTGATACCAGTACCAAAGTTGATAAT GCTATGTCAAGACTGTTGAAGAAGTATGATGTA	505
	TACATCATACTTCTTCAACAGTCTTGACATAGCATTATCAACTT TGGTACTGGTATCAAT**T**TCTTTTAGTAAGTTAAAGAATTTATGG ACACTACAAAGGAAAGAATAGAAAAAAGTAAAT	506
	CTAAAAGA**A**ATTGATAC	507
	GTATCAAT**T**TCTTTTAG	508
Retinoblastoma Glu137Term GAA-TAA	TGATTTACTTTTTTCTATTCTTTCCTTTGTAGTGTCCATAAATT CTTTAACTTACTAAAA**G**AAATTGATACCAGTACCAAAGTTGAT AATGCTATGTCAAGACTGTTGAAGAAGTATGATG	509

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CATCATACTTCTTCAACAGTCTTGACATAGCATTATCAACTTT GGTACTGGTATCAATTT**C**TTTTAGTAAGTTAAAGAATTTATGG ACACTACAAAGGAAAGAATAGAAAAAAGTAAATCA	510
	TACTAAAA**G**AAATTGAT	511
	ATCAATTT**C**TTTTAGTA	512
Retinoblastoma Gln176Term CAA-TAA	AAAATGTTAAAAAGTCATAATGTTTTTCTTTTCAGGACATGTGA ACTTATATATTTGACA**C**AACCCAGCAGTTCGTAAGTAGTTCAC AGAATGTTATTTTTCACTTAAAAAAAAAGATTTT	513
	AAAATCTTTTTTTTTAAGTGAAAAATAACATTCTGTGAACTACT TACGAACTGCTGGGTT**G**TGTCAAATATATAAGTTCACATGTCC TGAAAAGAAAAACATTATGACTTTTTAACATTTT	514
	ATTTGACA**C**AACCCAGC	515
	GCTGGGTT**G**TGTCAAAT	516
Retinoblastoma Ile185Thr ATA-ACA	TGATACATTTTTCCTGTTTTTTTTCTGCTTTCTATTTGTTTAATA GGATATCTACTGAAA**T**AAATTCTGCATTGGTGCTAAAAGTTTC TTGGATCACATTTTTATTAGCTAAAGGTAAGTT	517
	AACTTACCTTTAGCTAATAAAAATGTGATCCAAGAAACTTTTA GCACCAATGCAGAATTT**A**TTTCAGTAGATATCCTATTAAACAA ATAGAAAGCAGAAAAAAAACAGGAAAAATGTATCA	518
	TACTGAAA**T**AAATTCTG	519
	CAGAATTT**A**TTTCAGTA	520
Retinoblastoma Gln207Term CAA-TAA	AAAGATCTGAATCTCTAACTTTCTTTAAAAATGTACATTTTTTT TTCAGGGGAAGTATTA**C**AAATGGAAGATGATCTGGTGATTTC ATTTCAGTTAATGCTATGTGTCCTTGACTATTTTA	521
	TAAAATAGTCAAGGACACATAGCATTAACTGAAATGAAATCAC CAGATCATCTTCCATTT**G**TAATACTTCCCCTGAAAAAAAAATG TACATTTTTAAAGAAAGTTAGAGATTCAGATCTTT	522
	AAGTATTA**C**AAATGGAA	523
	TTCCATTT**G**TAATACTT	524
Retinoblastoma Arg251Term CGA to TGA	GTTCTTATCTAATTTACCACTTTTACAGAAACAGCTGTTATACC CATTAATGGTTCACCT**C**GAACACCCAGGCGAGGTCAGAACA GGAGTGCACGGATAGCAAAACAACTAGAAAATGATA	525
	TATCATTTTCTAGTTGTTTTGCTATCCGTGCACTCCTGTTCTG ACCTCGCCTGGGTGTTC**G**AGGTGAACCATTAATGGGTATAAC AGCTGTTTCTGTAAAAGTGGTAAATTAGATAAGAAC	526

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GTTCACCT**C**GAACACCC	527
	GGGTGTTC**G**AGGTGAAC	528
Retinoblastoma Arg255Term CGA to TGA	TTTACCACTTTTACAGAAACAGCTGTTATACCCATTAATGGTT CACCTCGAACACCCAGG**C**GAGGTCAGAACAGGAGTGCACG GATAGCAAAACAACTAGAAAATGATACAAGAATTATTG	529
	CAATAATTCTTGTATCATTTTCTAGTTGTTTTGCTATCCGTGCA CTCCTGTTCTGACCTC**G**CCTGGGTGTTCGAGGTGAACCATTA ATGGGTATAACAGCTGTTTCTGTAAAAGTGGTAAA	530
	CACCCAGG**C**GAGGTCAG	531
	CTGACCTC**G**CCTGGGTG	532
Retinoblastoma Gln266Term CAA to TAA	ATTAATGGTTCACCTCGAACACCCAGGCGAGGTCAGAACAG GAGTGCACGGATAGCAAAA**C**AACTAGAAAATGATACAAGAAT TATTGAAGTTCTCTGTAAAGAACATGAATGTAATATAG	533
	CTATATTACATTCATGTTCTTTACAGAGAACTTCAATAATTCTT GTATCATTTTCTAGTT**G**TTTTGCTATCCGTGCACTCCTGTTCT GACCTCGCCTGGGTGTTCGAGGTGAACCATTAAT	534
	TAGCAAAA**C**AACTAGAA	535
	TTCTAGTT**G**TTTTGCTA	536
Retinoblastoma Arg320Term CGA to TGA	TGACATGTAAAGGATAATTGTCAGTGACTTTTTTCTTTCAAGG TTGAAAATCTTTCTAAA**C**GATACGAAGAAATTTATCTTAAAAAT AAAGATCTAGATGCAAGATTATTTTTGGATCATG	537
	CATGATCCAAAAATAATCTTGCATCTAGATCTTTATTTTTAAGA TAAATTTCTTCGTATC**G**TTTAGAAAGATTTTCAACCTTGAAAGA AAAAAGTCACTGACAATTATCCTTTACATGTCA	538
	TTTCTAAA**C**GATACGAA	539
	TTCGTATC**G**TTTAGAAA	540
Retinoblastoma Gln354Term CAG to TAG	ACAAATTGTAAATTTTCAGTATGTGAATGACTTCACTTATTGTT ATTTAGTTTTGAAACA**C**AGAGAACACCACGAAAAAGTAACCTT GATGAAGAGGTGAATGTAATTCCTCCACACACTC	541
	GAGTGTGTGGAGGAATTACATTCACCTCTTCATCAAGGTTAC TTTTTCGTGGTGTTCTCT**G**TGTTTCAAAACTAAATAACAATAA GTGAAGTCATTCACATACTGAAAATTTACAATTTGT	542
	TTGAAACA**C**AGAGAACA	543
	TGTTCTCT**G**TGTTTCAA	544

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Arg358Gly CGA to GGA	TTTTCAGTATGTGAATGACTTCACTTATTGTTATTTAGTTTTGA AACACAGAGAACACCA**C**GAAAAAGTAACCTTGATGAAGAGGT GAATGTAATTCCTCCACACACTCCAGTTAGGTATG	545
	CATACCTAACTGGAGTGTGTGGAGGAATTACATTCACCTCTT CATCAAGGTTACTTTTTC**G**TGGTGTTCTCTGTGTTTCAAAACT AAATAACAATAAGTGAAGTCATTCACATACTGAAAA	546
	GAACACCA**C**GAAAAAGT	547
	ACTTTTTC**G**TGGTGTTC	548
Retinoblastoma Arg358Term CGA to TGA	TTTTCAGTATGTGAATGACTTCACTTATTGTTATTTAGTTTTGA AACACAGAGAACACCA**C**GAAAAAGTAACCTTGATGAAGAGGT GAATGTAATTCCTCCACACACTCCAGTTAGGTATG	549
	CATACCTAACTGGAGTGTGTGGAGGAATTACATTCACCTCTT CATCAAGGTTACTTTTTC**G**TGGTGTTCTCTGTGTTTCAAAACT AAATAACAATAAGTGAAGTCATTCACATACTGAAAA	550
	GAACACCA**C**GAAAAAGT	551
	ACTTTTTC**G**TGGTGTTC	552
Retinoblastoma Ser397Term TCA to TAA	CTGTTATGAACACTATCCAACAATTAATGATGATTTTAAATTCA GCAAGTGATCAACCTT**C**AGAAAATCTGATTTCCTATTTTAACG TAAGCCATATATGAAACATTATTTATTGTAATAT	553
	ATATTACAATAAATAATGTTTCATATATGGCTTACGTTAAAATA GGAAATCAGATTTTCT**G**AAGGTTGATCACTTGCTGAATTTAAA ATCATCATTAATTGTTGGATAGTGTTCATAACAG	554
	TCAACCTT**C**AGAAAATC	555
	GATTTTCT**G**AAGGTTGA	556
Retinoblastoma Arg445Term CGA to TGA	TTTCATAATTGTGATTTTCTAAAATAGCAGGCTCTTATTTTTCT TTTTGTTTGTTTGTAG**C**GATACAAACTTGGAGTTCGCTTGTAT TACCGAGTAATGGAATCCATGCTTAAATCAGTAA	557
	TTACTGATTTAAGCATGGATTCCATTACTCGGTAATACAAGCG AACTCCAAGTTTGTATC**G**CTACAAACAAACAAAAAGAAAAATA AGAGCCTGCTATTTTAGAAAATCACAATTATGAAA	558
	GTTTGTAG**C**GATACAAA	559
	TTTGTATC**G**CTACAAAC	560
Retinoblastoma Arg455Term CGA to TGA	GCTCTTATTTTTCTTTTTGTTTGTTTGTAGCGATACAAACTTGG AGTTCGCTTGTATTAC**C**GAGTAATGGAATCCATGCTTAAATCA GTAAGTTAAAAACAATATAAAAAAATTTCAGCCG	561

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CGGCTGAAATTTTTTTATATTGTTTTTAACTTACTGATTTAAGC ATGGATTCCATTACTC**G**GTAATACAAGCGAACTCCAAGTTTGT ATCGCTACAAACAAACAAAAAGAAAAATAAGAGC	562
	TGTATTAC**C**GAGTAATG	563
	CATTACTC**G**GTAATACA	564
Retinoblastoma Arg552Term CGA to TGA	ATCGAAAGTTTTATCAAAGCAGAAGGCAACTTGACAAGAGAA ATGATAAAACATTTAGAA**C**GATGTGAACATCGAATCATGGAAT CCCTTGCATGGCTCTCAGTAAGTAGCTAAATAATTG	565
	CAATTATTTAGCTACTTACTGAGAGCCATGCAAGGGATTCCAT GATTCGATGTTCACATC**G**TTCTAAATGTTTTATCATTTCTCTTG TCAAGTTGCCTTCTGCTTTGATAAAACTTTCGAT	566
	ATTTAGAA**C**GATGTGAA	567
	TTCACATC**G**TTCTAAAT	568
Retinoblastoma Cys553Term TGT to TGA	AAGTTTTATCAAAGCAGAAGGCAACTTGACAAGAGAAATGATA AAACATTTAGAACGATG**T**GAACATCGAATCATGGAATCCCTTG CATGGCTCTCAGTAAGTAGCTAAATAATTGAAGAA	569
	TTCTTCAATTATTTAGCTACTTACTGAGAGCCATGCAAGGGAT TCCATGATTCGATGTTC**A**CATCGTTCTAAATGTTTTATCATTTC TCTTGTCAAGTTGCCTTCTGCTTTGATAAAACTT	570
	GAACGATG**T**GAACATCG	571
	CGATGTTC**A**CATCGTTC	572
Retinoblastoma Glu554Term GAA to TAA	AGTTTTATCAAAGCAGAAGGCAACTTGACAAGAGAAATGATAA AACATTTAGAACGATGT**G**AACATCGAATCATGGAATCCCTTG CATGGCTCTCAGTAAGTAGCTAAATAATTGAAGAAA	573
	TTTCTTCAATTATTTAGCTACTTACTGAGAGCCATGCAAGGGA TTCCATGATTCGATGTT**C**ACATCGTTCTAAATGTTTTATCATTT CTCTTGTCAAGTTGCCTTCTGCTTTGATAAAACT	574
	AACGATGT**G**AACATCGA	575
	TCGATGTT**C**ACATCGTT	576
Retinoblastoma Ser567Leu TCA to TTA	TACCTGGGAAAATTATGCTTACTAATGTGGTTTTAATTTCATC ATGTTTCATATAGGATT**C**ACCTTTATTTGATCTTATTAAACAAT CAAAGGACCGAGAAGGACCAACTGATCACCTTGA	577
	TCAAGGTGATCAGTTGGTCCTTCTCGGTCCTTTGATTGTTTAA TAAGATCAAATAAAGGT**G**AATCCTATATGAAACATGATGAAAT TAAAACCACATTAGTAAGCATAATTTTCCCAGGTA	578

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATAGGATTCACCTTTAT	579
	ATAAAGGTGAATCCTAT	580
Retinoblastoma Gln575Term CAA to TAA	AATGTGGTTTTAATTTCATCATGTTTCATATAGGATTCACCTTT ATTTGATCTTATTAAACAATCAAAGGACCGAGAAGGACCAACT GATCACCTTGAATCTGCTTGTCCTCTTAATCTTC	581
	GAAGATTAAGAGGACAAGCAGATTCAAGGTGATCAGTTGGTC CTTCTCGGTCCTTTGATTGTTTAATAAGATCAAATAAAGGTGA ATCCTATATGAAACATGATGAAATTAAAACCACATT	582
	TTATTAAACAATCAAAG	583
	CTTTGATTGTTTAATAA	584
Retinoblastoma Arg579Term CGA to TGA	ATTTCATCATGTTTCATATAGGATTCACCTTTATTTGATCTTAT TAAACAATCAAAGGACCGAGAAGGACCAACTGATCACCTTGA ATCTGCTTGTCCTCTTAATCTTCCTCTCCAGAATA	585
	TATTCTGGAGAGGAAGATTAAGAGGACAAGCAGATTCAAGGT GATCAGTTGGTCCTTCTCGGTCCTTTGATTGTTTAATAAGATC AAATAAAGGTGAATCCTATATGAAACATGATGAAAT	586
	CAAAGGACCGAGAAGGA	587
	TCCTTCTCGGTCCTTTG	588
Retinoblastoma Glu580Term GAA to TAA	TCATCATGTTTCATATAGGATTCACCTTTATTTGATCTTATTAA ACAATCAAAGGACCGAGAAGGACCAACTGATCACCTTGAATC TGCTTGTCCTCTTAATCTTCCTCTCCAGAATAATC	589
	GATTATTCTGGAGAGGAAGATTAAGAGGACAAGCAGATTCAA GGTGATCAGTTGGTCCTTCTCGGTCCTTTGATTGTTTAATAAG ATCAAATAAAGGTGAATCCTATATGAAACATGATGA	590
	AGGACCGAGAAGGACCA	591
	TGGTCCTTCTCGGTCCT	592
Retinoblastoma Ser634Term TCA to TGA	AGAAAAAAGGTTCAACTACGCGTGTAAATTCTACTGCAAATG CAGAGACACAAGCAACCTCAGCCTTCCAGACCCAGAAGCCA TTGAAATCTACCTCTCTTTCACTGTTTTATAAAAAAGG	593
	CCTTTTTTATAAAACAGTGAAAGAGAGGTAGATTTCAATGGCT TCTGGGTCTGGAAGGCTGAGGTTGCTTGTGTCTCTGCATTTG CAGTAGAATTTACACGCGTAGTTGAACCTTTTTTCT	594
	AGCAACCTCAGCCTTCC	595
	GGAAGGCTGAGGTTGCT	596

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Ala635Pro GCC to CCC	AAAAAAGGTTCAACTACGCGTGTAAATTCTACTGCAAATGCA GAGACACAAGCAACCTCAGCCTTCCAGACCCAGAAGCCATT GAAATCTACCTCTCTTTCACTGTTTTATAAAAAAGGTT	597
	AACCTTTTTTATAAAACAGTGAAAGAGAGGTAGATTTCAATGG CTTCTGGGTCTGGAAGGCTGAGGTTGCTTGTGTCTCTGCATT TGCAGTAGAATTTACACGCGTAGTTGAACCTTTTTT	598
	CAACCTCAGCCTTCCAG	599
	CTGGAAGGCTGAGGTTG	600
Retinoblastoma Gln639Term CAG to TAG	ACTACGCGTGTAAATTCTACTGCAAATGCAGAGACACAAGCA ACCTCAGCCTTCCAGACCCAGAAGCCATTGAAATCTACCTCT CTTTCACTGTTTTATAAAAAAGGTTAGTAGATGATTA	601
	TAATCATCTACTAACCTTTTTTATAAAACAGTGAAAGAGAGGT AGATTTCAATGGCTTCTGGGTCTGGAAGGCTGAGGTTGCTTG TGTCTCTGCATTTGCAGTAGAATTTACACGCGTAGT	602
	TCCAGACCCAGAAGCCA	603
	TGGCTTCTGGGTCTGGA	604
Retinoblastoma Leu657Pro CTA to CCA	TTGTAATTCAAAATGAACAGTAAAAATGACTAATTTTTCTTATT CCCACAGTGTATCGGCTAGCCTATCTCCGGCTAAATACACTT TGTGAACGCCTTCTGTCTGAGCACCCAGAATTAGA	605
	TCTAATTCTGGGTGCTCAGACAGAAGGCGTTCACAAAGTGTA TTTAGCCGGAGATAGGCTAGCCGATACACTGTGGGAATAAG AAAAATTAGTCATTTTTACTGTTCATTTTGAATTACAA	606
	GTATCGGCTAGCCTATC	607
	GATAGGCTAGCCGATAC	608
Retinoblastoma Arg661Trp CGG to TGG	AATGAACAGTAAAAATGACTAATTTTTCTTATTCCCACAGTGTA TCGGCTAGCCTATCTCCGGCTAAATACACTTTGTGAACGCCT TCTGTCTGAGCACCCAGAATTAGAACATATCATCT	609
	AGATGATATGTTCTAATTCTGGGTGCTCAGACAGAAGGCGTT CACAAAGTGTATTTAGCCGGAGATAGGCTAGCCGATACACTG TGGGAATAAGAAAAATTAGTCATTTTTACTGTTCATT	610
	CCTATCTCCGGCTAAAT	611
	ATTTAGCCGGAGATAGG	612
Retinoblastoma Leu662Pro CTA to CCA	AACAGTAAAAATGACTAATTTTTCTTATTCCCACAGTGTATCG GCTAGCCTATCTCCGGCTAAATACACTTTGTGAACGCCTTCT GTCTGAGCACCCAGAATTAGAACATATCATCTGGAC	613

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GTCCAGATGATATGTTCTAATTCTGGGTGCTCAGACAGAAGG CGTTCACAAAGTGTATTTAGCCGGAGATAGGCTAGCCGATAC ACTGTGGGAATAAGAAAAATTAGTCATTTTTACTGTT	614
	TCTCCGGCTAAATACAC	615
	GTGTATTTAGCCGGAGA	616
Retinoblastoma Glu675Term GAA to TAA	TATCGGCTAGCCTATCTCCGGCTAAATACACTTTGTGAACGC CTTCTGTCTGAGCACCCAGAATTAGAACATATCATCTGGACC CTTTTCCAGCACACCCTGCAGAATGAGTATGAACTCA	617
	TGAGTTCATACTCATTCTGCAGGGTGTGCTGGAAAAGGGTCC AGATGATATGTTCTAATTCTGGGTGCTCAGACAGAAGGCGTT CACAAAGTGTATTTAGCCGGAGATAGGCTAGCCGATA	618
	AGCACCCAGAATTAGAA	619
	TTCTAATTCTGGGTGCT	620
Retinoblastoma Gln685Pro CAG to CCG	TTTGTGAACGCCTTCTGTCTGAGCACCCAGAATTAGAACATA TCATCTGGACCCTTTTCCAGCACACCCTGCAGAATGAGTATG AACTCATGAGAGACAGGCATTTGGACCAAGTAAGAAA	621
	TTTCTTACTTGGTCCAAATGCCTGTCTCTCATGAGTTCATACT CATTCTGCAGGGTGTGCTGGAAAAGGGTCCAGATGATATGTT CTAATTCTGGGTGCTCAGACAGAAGGCGTTCACAAA	622
	CCTTTTCCAGCACACCC	623
	GGGTGTGCTGGAAAAGG	624
Retinoblastoma Cys706Tyr TGT to TAT	AAAACCATGTAATAAAATTCTGACTACTTTTACATCAATTTATT TACTAGATTATGATGTGTTCCATGTATGGCATATGCAAAGTGA AGAATATAGACCTTAAATTCAAAATCATTGTAAC	625
	GTTACAATGATTTTGAATTTAAGGTCTATATTCTTCACTTTGCA TATGCCATACATGGAACACATCATAATCTAGTAAATAAATTGA TGTAAAAGTAGTCAGAATTTTATTACATGGTTTT	626
	TATGATGTGTTCCATGT	627
	ACATGGAACACATCATA	628
Retinoblastoma Cys712Arg TGC to CGC	TTCTGACTACTTTTACATCAATTTATTTACTAGATTATGATGTG TTCCATGTATGGCATATGCAAAGTGAAGAATATAGACCTTAAA TTCAAAATCATTGTAACAGCATACAAGGATCTTC	629
	GAAGATCCTTGTATGCTGTTACAATGATTTTGAATTTAAGGTC TATATTCTTCACTTTGCATATGCCATACATGGAACACATCATA ATCTAGTAAATAAATTGATGTAAAAGTAGTCAGAA	630

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATGGCATATGCAAAGTG	631
	CACTTTGCATATGCCAT	632
Retinoblastoma Tyr728Term TAC to TAA	GTATGGCATATGCAAAGTGAAGAATATAGACCTTAAATTCAAA ATCATTGTAACAGCATACAAGGATCTTCCTCATGCTGTTCAG GAGGTAGGTAATTTTCCATAGTAAGTTTTTTTGATA	633
	TATCAAAAAAACTTACTATGGAAAATTACCTACCTCCTGAACA GCATGAGGAAGATCCTTGTATGCTGTTACAATGATTTTGAATT TAAGGTCTATATTCTTCACTTTGCATATGCCATAC	634
	ACAGCATACAAGGATCT	635
	AGATCCTTGTATGCTGT	636
Retinoblastoma Glu748Term GAG to TAG	TTTTTTTTTTTTTTTACTGTTCTTCCTCAGACATTCAAACGTGT TTTGATCAAAGAAGAGGAGTATGATTCTATTATAGTATTCTATA ACTCGGTCTTCATGCAGAGACTGAAAACAAATA	637
	TATTTGTTTTCAGTCTCTGCATGAAGACCGAGTTATAGAATAC TATAATAGAATCATACTCCTCTTCTTTGATCAAAACACGTTTGA ATGTCTGAGGAAGAACAGTAAAAAAAAAAAAAAA	638
	AAGAAGAGGAGTATGAT	639
	ATCATACTCCTCTTCTT	640
Retinoblastoma Gln762Term CAG to TAG	GTTTTGATCAAAGAAGAGGAGTATGATTCTATTATAGTATTCT ATAACTCGGTCTTCATGCAGAGACTGAAAACAAATATTTTGCA GTATGCTTCCACCAGGGTAGGTCAAAAGTATCCTT	641
	AAGGATACTTTTGACCTACCCTGGTGGAAGCATACTGCAAAA TATTTGTTTTCAGTCTCTGCATGAAGACCGAGTTATAGAATAC TATAATAGAATCATACTCCTCTTCTTTGATCAAAAC	642
	TCTTCATGCAGAGACTG	643
	CAGTCTCTGCATGAAGA	644
Retinoblastoma Arg787Term CGA-TGA	TAATCTACTTTTTTGTTTTTGCTCTAGCCCCCTACCTTGTCAC CAATACCTCACATTCCTCGAAGCCCTTACAAGTTTCCTAGTTC ACCCTTACGGATTCCTGGAGGGAACATCTATATTT	645
	AAATATAGATGTTCCCTCCAGGAATCCGTAAGGGTGAACTAG GAAACTTGTAAGGGCTTCGAGGAATGTGAGGTATTGGTGACA AGGTAGGGGGCTAGAGCAAAAACAAAAAAGTAGATTA	646
	ACATTCCTCGAAGCCCT	647
	AGGGCTTCGAGGAATGT	648

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Ser816Term TCA to TGA	CCTTACGGATTCCTGGAGGGAACATCTATATTTCACCCCTGA AGAGTCCATATAAAATTT**C**AGAAGGTCTGCCAACACCAACAA AAATGACTCCAAGATCAAGGTGTGTGTTTTCTCTTTA	649
	TAAAGAGAAAACACACACCTTGATCTTGGAGTCATTTTTGTTG GTGTTGGCAGACCTTCT**G**AAATTTTATATGGACTCTTCAGGG GTGAAATATAGATGTTCCCTCCAGGAATCCGTAAGG	650
	TAAAATTT**C**AGAAGGTC	651
	GACCTTCT**G**AAATTTTA	652

EXAMPLE 8

BRCA1 and BRCA2

Breast cancer is the second major cause of cancer death in American women, with an estimated 44,190 lives lost (290 men and 43,900 women) in the US in 1997. While ovarian cancer accounts for fewer deaths than breast cancer, it still represents 4% of all female cancers. In 1994, two breast cancer susceptibility genes were identified: BRCA1 on chromosome 17 and BRCA2 on chromosome 13. When a woman carries a mutation in either BRCA1 or BRCA2, she is at increased risk of being diagnosed with breast or ovarian cancer at some point in her life.

Ford *et al.*, *Am. J. Hum. Genet.* 62: 676-689 (1998) assessed the contribution of BRCA1 and BRCA2 to inherited breast cancer by linkage and mutation analysis in 237 families, each with at least 4 cases of breast cancer. Families were included without regard to the occurrence of ovarian or other cancers. Overall, disease was linked to BRCA1 in an estimated 52% of families, to BRCA2 in 32% of families, and to neither gene in 16%, suggesting other predisposition genes. The majority (81%) of the breast-ovarian cancer families were due to BRCA1, with most others (14%) due to BRCA2. Conversely, the majority (76%) of families with both male and female breast cancer were due to BRCA2. The largest proportion (67%) of families due to other genes were families with 4 or 5 cases of female breast cancer only.

More than 75% of the reported mutations in the BRCA1 gene result in truncated proteins. Couch *et al.*, *Hum. Mutat.* 8: 8-18, 1996. (1996) reported a total of 254 BRCA1 mutations, 132 (52%) of which were unique. A total of 221 (87%) of all mutations or 107 (81%) of the unique mutations are small deletions, insertions, nonsense point mutations, splice variants, and regulatory mutations that result in

truncation or absence of the BRCA1 protein. A total of 11 disease-associated missense mutations (5 unique) and 21 variants (19 unique) as yet unclassified as missense mutations or polymorphisms had been detected. Thirty-five independent benign polymorphisms had been described. The most common mutations were 185delAG and 5382insC, which accounted for 30 (11.7%) and 26 (10.1%), respectively, of all the mutations.

Most BRCA2 mutations are predicted to result in a truncated protein product. The smallest known cancer-associated deletion removes from the C terminus only 224 of the 3,418 residues constituting BRCA2, suggesting that these terminal amino acids are critical for BRCA2 function. Studies (Spain *et al.*, Proc. Natl. Acad. Sci. 96:13920-13925 (1999)) suggest that such truncations eliminate or interfere with 2 nuclear localization signals that reside within the final 156 residues of BRCA2, suggesting that the vast majority of BRCA2 mutants are nonfunctional because they are not translocated into the nucleus.

The attached table discloses the correcting oligonucleotide base sequences for the BRACA1 and BRACA2 oligonucleotides of the invention.

Table 14
BRCA1 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Met-1-Ile ATG to ATT	CTGCGCTCAGGAGGCCTTCACCCTCTGCTCTGGGTAAAGTT CATTGGAACAGAAAGAAATGGATTTATCTGCTCTTCGCGTTG AAGAAGTACAAAATGTCATTAATGCTATGCAGAAAATC	653
	GATTTTCTGCATAGCATTAATGACATTTTGTACTTCTTCAACG CGAAGAGCAGATAAATCCATTTCTTTCTGTTCCAATGAACTTT ACCCAGAGCAGAGGGTGAAGGCCTCCTGAGCGCAG	654
	AAAGAAATGGATTTATC	655
	GATAAATCCATTTCTTT	656
Breast Cancer Val-11-Ala GTA to GCA	CTGGGTAAAGTTCATTGGAACAGAAAGAAATGGATTTATCTG CTCTTCGCGTTGAAGAAGTACAAAATGTCATTAATGCTATGCA GAAAATCTTAGAGTGTCCCATCTGTCTGGAGTTGAT	657
	ATCAACTCCAGACAGATGGGACACTCTAAGATTTTCTGCATA GCATTAATGACATTTTGTACTTCTTCAACGCGAAGAGCAGATA AATCCATTTCT. TCTGTTCCAATGAACTTTACCCAG	658
	TGAAGAAGTACAAAATG	659

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CATTTTGTACTTCTTCA	660
Breast Cancer Ile-21-Val ATC to GTC	ATGGATTTATCTGCTCTTCGCGTTGAAGAAGTACAAAATGTCA TTAATGCTATGCAGAAAATCTTAGAGTGTCCCATCTGTCTGG AGTTGATCAAGGAACCTGTCTCCACAAAGTGTGACC	661
	GGTCACACTTTGTGGAGACAGGTTCCTTGATCAACTCCAGAC AGATGGGACACTCTAAGATTTTCTGCATAGCATTAATGACATT TTGTACTTCTTCAACGCGAAGAGCAGATAAATCCAT	662
	TGCAGAAAATCTTAGAG	663
	CTCTAAGATTTTCTGCA	664
Breast Cancer Leu-22-Ser TTA to TCA	ATTTATCTGCTCTTCGCGTTGAAGAAGTACAAAATGTCATTAA TGCTATGCAGAAAATCTTAGAGTGTCCCATCTGTCTGGAGTT GATCAAGGAACCTGTCTCCACAAAGTGTGACCACAT	665
	ATGTGGTCACACTTTGTGGAGACAGGTTCCTTGATCAACTCC AGACAGATGGGACACTCTAAGATTTTCTGCATAGCATTAATG ACATTTTGTACTTCTTCAACGCGAAGAGCAGATAAAT	666
	GAAAATCTTAGAGTGTC	667
	GACACTCTAAGATTTTC	668
Breast Cancer Cys-39-Tyr TGT to TAT	AGAAAATCTTAGAGTGTCCCATCTGTCTGGAGTTGATCAAGG AACCTGTCTCCACAAAGTGTGACCACATATTTTGCAAATTTTG CATGCTGAAACTTCTCAACCAGAAGAAAGGGCCTTC	669
	GAAGGCCCTTTCTTCTGGTTGAGAAGTTTCAGCATGCAAAAT TTGCAAAATATGTGGTCACACTTTGTGGAGACAGGTTCCTTG ATCAACTCCAGACAGATGGGACACTCTAAGATTTTCT	670
	CACAAAGTGTGACCACA	671
	TGTGGTCACACTTTGTG	672
Breast Cancer Cys-61-Gly TGT to GGT	CACATATTTTGCAAATTTTGCATGCTGAAACTTCTCAACCAGA AGAAAGGGCCTTCACAGTGTCCTTTATGTAAGAATGATATAAC CAAAAGGAGCCTACAAGAAAGTACGAGATTTAGTC	673
	GACTAAATCTCGTACTTTCTTGTAGGCTCCTTTTGGTTATATC ATTCTTACATAAAGGACACTGTGAAGGCCCTTTCTTCTGGTT GAGAAGTTTCAGCATGCAAAATTTGCAAAATATGTG	674
	CTTCACAGTGTCCTTTA	675
	TAAAGGACACTGTGAAG	676

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Leu-63-Stop TTA to TAA	TTTGCAAATTTTGCATGCTGAAACTTCTCAACCAGAAGAAAGG GCCTTCACAGTGTCCTTTATGTAAGAATGATATAACCAAAAGG AGCCTACAAGAAAGTACGAGATTTAGTCAACTTGT	677
	ACAAGTTGACTAAATCTCGTACTTTCTTGTAGGCTCCTTTTGG TTATATCATTCTTACATAAAGGACACTGTGAAGGCCCTTTCTT CTGGTTGAGAAGTTTCAGCATGCAAAATTTGCAAA	678
	GTGTCCTTTATGTAAGA	679
	TCTTACATAAAGGACAC	680
Breast Cancer Cys-64-Arg TGT to CGT	TGCAAATTTTGCATGCTGAAACTTCTCAACCAGAAGAAAGGG CCTTCACAGTGTCCTTTATGTAAGAATGATATAACCAAAAGGA GCCTACAAGAAAGTACGAGATTTAGTCAACTTGTTG	681
Breast Cancer Cys-64-Gly TGT to GGT	CAACAAGTTGACTAAATCTCGTACTTTCTTGTAGGCTCCTTTT GGTTATATCATTCTTACATAAAGGACACTGTGAAGGCCCTTTC TTCTGGTTGAGAAGTTTCAGCATGCAAAATTTGCA	682
	GTCCTTTATGTAAGAAT	683
	ATTCTTACATAAAGGAC	684
Breast Cancer Cys-64-Tyr TGT to TAT	GCAAATTTTGCATGCTGAAACTTCTCAACCAGAAGAAAGGGC CTTCACAGTGTCCTTTATGTAAGAATGATATAACCAAAAGGAG CCTACAAGAAAGTACGAGATTTAGTCAACTTGTTGA	685
	TCAACAAGTTGACTAAATCTCGTACTTTCTTGTAGGCTCCTTT TGGTTATATCATTCTTACATAAAGGACACTGTGAAGGCCCTTT CTTCTGGTTGAGAAGTTTCAGCATGCAAAATTTGC	686
	TCCTTTATGTAAGAATG	687
	CATTCTTACATAAAGGA	688
Breast Cancer Gln-74-Stop CAA to TAA	CAGAAGAAAGGGCCTTCACAGTGTCCTTTATGTAAGAATGAT ATAACCAAAAGGAGCCTACAAGAAAGTACGAGATTTAGTCAA CTTGTTGAAGAGCTATTGAAAATCATTTGTGCTTTTC	689
	GAAAAGCACAAATGATTTTCAATAGCTCTTCAACAAGTTGACT AAATCTCGTACTTTCTTGTAGGCTCCTTTTGGTTATATCATTCT TACATAAAGGACACTGTGAAGGCCCTTTCTTCTG	690
	GGAGCCTACAAGAAAGT	691
	ACTTTCTTGTAGGCTCC	692

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Tyr-105-Cys TAT to TGT	AGCTATTGAAAATCATTTGTGCTTTTCAGCTTGACACAGGTTT GGAGTATGCAAACAGCTATAATTTTGCAAAAAAGGAAAATAAC TCTCCTGAACATCTAAAAGATGAAGTTTCTATCAT	693
	ATGATAGAAACTTCATCTTTTAGATGTTCAGGAGAGTTATTTT CCTTTTTTGCAAAATTATAGCTGTTTGCATACTCCAAACCTGT GTCAAGCTGAAAAGCACAAATGATTTTCAATAGCT	694
	AAACAGCTATAATTTTG	695
	CAAAATTATAGCTGTTT	696
Breast Cancer Asn-158-Tyr AAC to TAC	CTACAGAGTGAACCCGAAAATCCTTCCTTGCAGGAAACCAGT CTCAGTGTCCAACTCTCTAACCTTGGAACTGTGAGAACTCTG AGGACAAAGCAGCGGATACAACCTCAAAAGACGTCTG	697
	CAGACGTCTTTTGAGGTTGTATCCGCTGCTTTGTCCTCAGAG TTCTCACAGTTCCAAGGTTAGAGAGTTGGACACTGAGACTGG TTTCCTGCAAGGAAGGATTTTCGGGTTCACTCTGTAG	698
	AACTCTCTAACCTTGGA	699
	TCCAAGGTTAGAGAGTT	700
Breast Cancer Gln-169-Stop CAG to TAG	GAAACCAGTCTCAGTGTCCAACTCTCTAACCTTGGAACTGTG AGAACTCTGAGGACAAAGCAGCGGATACAACCTCAAAAGAC GTCTGTCTACATTGAATTGGGATCTGATTCTTCTGAAG	701
	CTTCAGAAGAATCAGATCCCAATTCAATGTAGACAGACGTCTT TTGAGGTTGTATCCGCTGCTTTGTCCTCAGAGTTCTCACAGT TCCAAGGTTAGAGAGTTGGACACTGAGACTGGTTTC	702
	GGACAAAGCAGCGGATA	703
	TATCCGCTGCTTTGTCC	704
Breast Cancer Trp-353-Stop TGG to TAG	CTCCCAGCACAGAAAAAAAGGTAGATCTGAATGCTGATCCCC TGTGTGAGAGAAAAGAATGGAATAAGCAGAAACTGCCATGCT CAGAGAATCCTAGAGATACTGAAGATGTTCCTTGGAT	705
	ATCCAAGGAACATCTTCAGTATCTCTAGGATTCTCTGAGCAT GGCAGTTTCTGCTTATTCCATTCTTTTCTCTCACACAGGGGAT CAGCATTCAGATCTACCTTTTTTTCTGTGCTGGGAG	706
	AAAAGAATGGAATAAGC	707
	GCTTATTCCATTCTTTT	708
Breast Cancer Ile-379-Met ATT to ATG	ATGCTCAGAGAATCCTAGAGATACTGAAGATGTTCCTTGGAT AACACTAAATAGCAGCATTCAGAAAGTTAATGAGTGGTTTTCC AGAAGTGATGAACTGTTAGGTTCTGATGACTCACAT	709

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATGTGAGTCATCAGAACCTAACAGTTCATCACTTCTGGAAAAC CACTCATTAACTTTCTGAATGCTGCTATTTAGTGTTATCCAAG GAACATCTTCAGTATCTCTAGGATTCTCTGAGCAT	710
	AGCAGCATTCAGAAAGT	711
	ACTTTCTGAATGCTGCT	712
Breast Cancer Glu-421-Gly GAA to GGA	GGGAGTCTGAATCAAATGCCAAAGTAGCTGATGTATTGGACG TTCTAAATGAGGTAGATGAATATTCTGGTTCTTCAGAGAAAAT AGACTTACTGGCCAGTGATCCTCATGAGGCTTTAAT	713
	ATTAAAGCCTCATGAGGATCACTGGCCAGTAAGTCTATTTTCT CTGAAGAACCAGAATATTCATCTACCTCATTTAGAACGTCCAA TACATCAGCTACTTTGGCATTTGATTCAGACTCCC	714
	GGTAGATGAATATTCTG	715
	CAGAATATTCATCTACC	716
Breast Cancer Phe-461-Leu TTT to CTT	ATATGTAAAAGTGAAAGAGTTCACTCCAAATCAGTAGAGAGTA ATATTGAAGACAAAATATTTGGGAAAACCTATCGGAAGAAGG CAAGCCTCCCCAACTTAAGCCATGTAACTGAAAATC	717
	GATTTTCAGTTACATGGCTTAAGTTGGGGAGGCTTGCCTTCT TCCGATAGGTTTTCCCAAATATTTTGTCTTCAATATTACTCTCT ACTGATTTGGAGTGAACTCTTTCACTTTTACATAT	718
	ACAAAATATTTGGGAAA	719
	TTTCCCAAATATTTTGT	720
Breast Cancer Tyr-465-Leu TAT to GAT	GAAAGAGTTCACTCCAAATCAGTAGAGAGTAATATTGAAGAC AAAATATTTGGGAAAACCTATCGGAAGAAGGCAAGCCTCCCC AACTTAAGCCATGTAACTGAAAATCTAATTATAGGAG	721
	CTCCTATAATTAGATTTTCAGTTACATGGCTTAAGTTGGGGAG GCTTGCCTTCTTCCGATAGGTTTTCCCAAATATTTTGTCTTCA ATATTACTCTCTACTGATTTGGAGTGAACTCTTTC	722
	GGAAAACCTATCGGAAG	723
	CTTCCGATAGGTTTTCC	724
Breast Cancer Gly-484-Stop GGA to TGA	ACCTATCGGAAGAAGGCAAGCCTCCCCAACTTAAGCCATGTA ACTGAAAATCTAATTATAGGAGCATTTGTTACTGAGCCACAGA TAATACAAGAGCGTCCCCTCACAAATAAATTAAAGC	725
	GCTTTAATTTATTTGTGAGGGGACGCTCTTGTATTATCTGTGG CTCAGTAACAAATGCTCCTATAATTAGATTTTCAGTTACATGG CTTAAGTTGGGGAGGCTTGCCTTCTTCCGATAGGT	726

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAATTATAGGAGCATTT	727
	AAATGCTCCTATAATTA	728
Breast Cancer Arg-507-Ile AGA to ATA	TTACTGAGCCACAGATAATACAAGAGCGTCCCCTCACAAATA AATTAAAGCGTAAAAGGAGACCTACATCAGGCCTTCATCCTG AGGATTTTATCAAGAAAGCAGATTTGGCAGTTCAAAA	729
	TTTTGAACTGCCAAATCTGCTTTCTTGATAAAATCCTCAGGAT GAAGGCCTGATGTAGGTCTCCTTTTACGCTTTAATTTATTTGT GAGGGGACGCTCTTGTATTATCTGTGGCTCAGTAA	730
	TAAAAGGAGACCTACAT	731
	ATGTAGGTCTCCTTTTA	732
Breast Cancer Ser-510-Stop TCA to TGA	CACAGATAATACAAGAGCGTCCCCTCACAAATAAATTAAAGC GTAAAAGGAGACCTACATCAGGCCTTCATCCTGAGGATTTTA TCAAGAAAGCAGATTTGGCAGTTCAAAAGACTCCTGA	733
	TCAGGAGTCTTTTGAACTGCCAAATCTGCTTTCTTGATAAAAT CCTCAGGATGAAGGCCTGATGTAGGTCTCCTTTTACGCTTTA ATTTATTTGTGAGGGGACGCTCTTGTATTATCTGTG	734
	ACCTACATCAGGCCTTC	735
	GAAGGCCTGATGTAGGT	736
Breast Cancer Gln-526-Stop CAA to TAA	AGGAGACCTACATCAGGCCTTCATCCTGAGGATTTTATCAAG AAAGCAGATTTGGCAGTTCAAAAGACTCCTGAAATGATAAATC AGGGAACTAACCAAACGGAGCAGAATGGTCAAGTGA	737
	TCACTTGACCATTCTGCTCCGTTTGGTTAGTTCCCTGATTTAT CATTTCAGGAGTCTTTTGAACTGCCAAATCTGCTTTCTTGATA AAATCCTCAGGATGAAGGCCTGATGTAGGTCTCCT	738
	TGGCAGTTCAAAAGACT	739
	AGTCTTTTGAACTGCCA	740
Breast Cancer Gln-541-Stop CAG to TAG	AGGAGACCTACATCAGGCCTTCATCCTGAGGATTTTATCAAG AAAGCAGATTTGGCAGTTCAAAAGACTCCTGAAATGATAAATC AGGGAACTAACCAAACGGAGCAGAATGGTCAAGTGA	741
	TCACTTGACCATTCTGCTCCGTTTGGTTAGTTCCCTGATTTAT CATTTCAGGAGTCTTTTGAACTGCCAAATCTGCTTTCTTGATA AAATCCTCAGGATGAAGGCCTGATGTAGGTCTCCT	742
	AAACGGAGCAGAATGGT	743
	ACCATTCTGCTCCGTTT	744

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Gly-552-Val GGT to GTT	TAAATCAGGGAACTAACCAAACGGAGCAGAATGGTCAAGTGA TGAATATTACTAATAGTGGTCATGAGAATAAAACAAAAGGTGA TTCTATTCAGAATGAGAAAAATCCTAACCCAATAGA	745
	TCTATTGGGTTAGGATTTTTCTCATTCTGAATAGAATCACCTTT TGTTTTATTCTCATGACCACTATTAGTAATATTCATCACTTGAC CATTCTGCTCCGTTTGGTTAGTTCCCTGATTTA	746
	TAATAGTGGTCATGAGA	747
	TCTCATGACCACTATTA	748
Breast Cancer Gln-563-Stop CAG to TAG	GGTCAAGTGATGAATATTACTAATAGTGGTCATGAGAATAAAA CAAAAGGTGATTCTATTCAGAATGAGAAAAATCCTAACCCAAT AGAATCACTCGAAAAAGAATCTGCTTTCAAAACGA	749
	TCGTTTTGAAAGCAGATTCTTTTTCGAGTGATTCTATTGGGTT AGGATTTTTCTCATTCTGAATAGAATCACCTTTTGTTTTATTCT CATGACCACTATTAGTAATATTCATCACTTGACC	750
	ATTCTATTCAGAATGAG	751
	CTCATTCTGAATAGAAT	752
Ovarian Cancer Lys-607-Stop AAA to TAA	ATAAGCAGCAGTATAAGCAATATGGAACTCGAATTAAATATCC ACAATTCAAAAGCACCTAAAAAAGAATAGGCTGAGGAGGAAGT CTTCTACCAGGCATATTCATGCGCTTGAACTAGTAG	753
	CTACTAGTTCAAGCGCATGAATATGCCTGGTAGAAGACTTCC TCCTCAGCCTATTCTTTTTAGGTGCTTTTGAATTGTGGATATT TAATTCGAGTTCCATATTGCTTATACTGCTGCTTAT	754
	AAGCACCTAAAAAGAAT	755
	ATTCTTTTTAGGTGCTT	756
Breast Cancer Leu-639-Stop TTG to TAG	ATATTCATGCGCTTGAACTAGTAGTCAGTAGAAATCTAAGCCC ACCTAATTGTACTGAATTGCAAATTGATAGTTGTTCTAGCAGT GAAGAGATAAAGAAAAAAAAGTACAACCAAATGCC	757
	GGCATTTGGTTGTACTTTTTTTTCTTTATCTCTTCACTGCTAGA ACAACTATCAATTTGCAATTCAGTACAATTAGGTGGGCTTAGA TTTCTACTGACTACTAGTTCAAGCGCATGAATAT	758
	TACTGAATTGCAAATTG	759
	CAATTTGCAATTCAGTA	760
Breast Cancer Asp-693-Asn GAC to AAC	GAACCTGCAACTGGAGCCAAGAAGAGTAACAAGCCAAATGAA CAGACAAGTAAAAGACATGACAGCGATACTTTCCCAGAGCTG AAGTTAACAAATGCACCTGGTTCTTTTACTAAGTGTT	761

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AACACTTAGTAAAAGAACCAGGTGCATTTGTTAACTTCAGCTC TGGGAAAGTATCGCTGTCATGTCTTTTACTTGTCTGTTCATTT GGCTTGTTACTCTTCTTGGCTCCAGTTGCAGGTTC	762
	AAAGACATGACAGCGAT	763
	ATCGCTGTCATGTCTTT	764
Ovarian Cancer Glu-720-Stop GAA to TAA	CTGAAGTTAACAAATGCACCTGGTTCTTTTACTAAGTGTTCAA ATACCAGTGAACTTAAAGAATTTGTCAATCCTAGCCTTCCAAG AGAAGAAAAAGAAGAGAAACTAGAAACAGTTAAAG	765
	CTTTAACTGTTTCTAGTTTCTCTTCTTTTTCTTCTCTTGGAAGG CTAGGATTGACAAATTCTTTAAGTTCACTGGTATTTGAACACT TAGTAAAAGAACCAGGTGCATTTGTTAACTTCAG	766
	AACTTAAAGAATTTGTC	767
	GACAAATTCTTTAAGTT	768
Breast Cancer Glu-755-Stop GAA to TAA	CTAGAAACAGTTAAAGTGTCTAATAATGCTGAAGACCCCAAA GATCTCATGTTAAGTGGAGAAAGGGTTTTGCAAACTGAAAGA TCTGTAGAGAGTAGCAGTATTTCATTGGTACCTGGTA	769
	TACCAGGTACCAATGAAATACTGCTACTCTCTACAGATCTTTC AGTTTGCAAAACCCTTTCTCCACTTAACATGAGATCTTTGGGG TCTTCAGCATTATTAGACACTTTAACTGTTTCTAG	770
	TAAGTGGAGAAAGGGTT	771
	AACCCTTTCTCCACTTA	772
Breast Cancer Ser-770-Stop TCA to TAA	TCATGTTAAGTGGAGAAAGGGTTTTGCAAACTGAAAGATCTG TAGAGAGTAGCAGTATTTCATTGGTACCTGGTACTGATTATG GCACTCAGGAAAGTATCTCGTTACTGGAAGTTAGCAC	773
	GTGCTAACTTCCAGTAACGAGATACTTTCCTGAGTGCCATAA TCAGTACCAGGTACCAATGAAATACTGCTACTCTCTACAGAT CTTTCAGTTTGCAAAACCCTTTCTCCACTTAACATGA	774
	CAGTATTTCATTGGTAC	775
	GTACCAATGAAATACTG	776
Breast Cancer Val-772-Ala GTA to GCA	TAAGTGGAGAAAGGGTTTTGCAAACTGAAAGATCTGTAGAGA GTAGCAGTATTTCATTGGTACCTGGTACTGATTATGGCACTC AGGAAAGTATCTCGTTACTGGAAGTTAGCACTCTAGG	777
	CCTAGAGTGCTAACTTCCAGTAACGAGATACTTTCCTGAGTG CCATAATCAGTACCAGGTACCAATGAAATACTGCTACTCTCTA CAGATCTTTCAGTTTGCAAAACCCTTTCTCCACTTA	778

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTCATTGGTACCTGGTA	779
	TACCAGGTACCAATGAA	780
Breast Cancer Gln-780-Stop CAG to TAG	ACTGAAAGATCTGTAGAGAGTAGCAGTATTTCATTGGTACCT GGTACTGATTATGGCACTCAGGAAAGTATCTCGTTACTGGAA GTTAGCACTCTAGGGAAGGCAAAAACAGAACCAAATA	781
	TATTTGGTTCTGTTTTTGCCTTCCCTAGAGTGCTAACTTCCAG TAACGAGATACTTTCCTGAGTGCCATAATCAGTACCAGGTAC CAATGAAATACTGCTACTCTCTACAGATCTTTCAGT	782
	ATGGCACTCAGGAAAGT	783
	ACTTTCCTGAGTGCCAT	784
Breast Cancer Glu-797-Stop GAA to TAA	TATGGCACTCAGGAAAGTATCTCGTTACTGGAAGTTAGCACT CTAGGGAAGGCAAAAACAGAACCAAATAAATGTGTGAGTCAG TGTGCAGCATTTGAAAACCCCAAGGGACTAATTCATG	785
	CATGAATTAGTCCCTTGGGGTTTTCAAATGCTGCACACTGAC TCACACATTTATTTGGTTCTGTTTTTGCCTTCCCTAGAGTGCT AACTTCCAGTAACGAGATACTTTCCTGAGTGCCATA	786
	CAAAAACAGAACCAAAT	787
	ATTTGGTTCTGTTTTTG	788
Breast Cancer Lys-820-Glu AAA to GAA	AAATGTGTGAGTCAGTGTGCAGCATTTGAAAACCCCAAGGGA CTAATTCATGGTTGTTCCAAAGATAATAGAAATGACACAGAAG GCTTTAAGTATCCATTGGGACATGAAGTTAACCACA	789
	TGTGGTTAACTTCATGTCCCAATGGATACTTAAAGCCTTCTGT GTCATTTCTATTATCTTTGGAACAACCATGAATTAGTCCCTTG GGGTTTTCAAATGCTGCACACTGACTCACACATTT	790
	GTTGTTCCAAAGATAAT	791
	ATTATCTTTGGAACAAC	792
Breast Cancer Thr-826-Lys ACA to AAA	CAGCATTTGAAAACCCCAAGGGACTAATTCATGGTTGTTCCA AAGATAATAGAAATGACACAGAAGGCTTTAAGTATCCATTGG GACATGAAGTTAACCACAGTCGGGAAACAAGCATAGA	793
	TCTATGCTTGTTTCCCGACTGTGGTTAACTTCATGTCCCAATG GATACTTAAAGCCTTCTGTGTCATTTCTATTATCTTTGGAACA ACCATGAATTAGTCCCTTGGGGTTTTCAAATGCTG	794
	AAATGACACAGAAGGCT	795
	AGCCTTCTGTGTCATTT	796

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Arg-841-Trp CGG to TGG	GATAATAGAAATGACACAGAAGGCTTTAAGTATCCATTGGGA CATGAAGTTAACCACAGT**C**GGGAAACAAGCATAGAAATGGAA GAAAGTGAACTTGATGCTCAGTATTTGCAGAATACAT	797
	ATGTATTCTGCAAATACTGAGCATCAAGTTCACTTTCTTCCAT TTCTATGCTTGTTTCCC**G**ACTGTGGTTAACTTCATGTCCCAAT GGATACTTAAAGCCTTCTGTGTCATTTCTATTATC	798
	ACCACAGT**C**GGGAAACA	799
	TGTTTCCC**G**ACTGTGGT	800
Breast Cancer Pro-871-Leu CCG to CTG	AACTTGATGCTCAGTATTTGCAGAATACATTCAAGGTTTCAAA GCGCCAGTCATTTGCTC**C**GTTTTCAAATCCAGGAAATGCAGA AGAGGAATGTGCAACATTCTCTGCCCACTCTGGGTC	801
	GACCCAGAGTGGGCAGAGAATGTTGCACATTCCTCTTCTGCA TTTCCTGGATTTGAAAAC**G**GAGCAAATGACTGGCGCTTTGAA ACCTTGAATGTATTCTGCAAATACTGAGCATCAAGTT	802
	ATTTGCTC**C**GTTTTCAA	803
	TTGAAAAC**G**GAGCAAAT	804
Breast Cancer Leu-892-Ser TTA to TCA	TTTCAAATCCAGGAAATGCAGAAGAGGAATGTGCAACATTCT CTGCCCACTCTGGGTCCT**T**AAAGAAACAAAGTCCAAAAGTCA CTTTTGAATGTGAACAAAAGGAAGAAAATCAAGGAAA	805
	TTTCCTTGATTTTCTTCCTTTTGTTCACATTCAAAAGTGACTTT TGGACTTTGTTTCTTT**A**AGGACCCAGAGTGGGCAGAGAATGT TGCACATTCCTCTTCTGCATTTCCTGGATTTGAAA	806
	TGGGTCCT**T**AAAGAAAC	807
	GTTTCTTT**A**AGGACCCA	808
Breast Cancer Glu-908-Stop GAA to TAA	CACTCTGGGTCCTTAAAGAAACAAAGTCCAAAAGTCACTTTTG AATGTGAACAAAAGGAA**G**AAAATCAAGGAAAGAATGAGTCTA ATATCAAGCCTGTACAGACAGTTAATATCACTGCAG	809
	CTGCAGTGATATTAACTGTCTGTACAGGCTTGATATTAGACTC ATTCTTTCCTTGATTTT**C**TTCCTTTTGTTCACATTCAAAAGTGA CTTTTGGACTTTGTTTCTTTAAGGACCCAGAGTG	810
	AAAAGGAA**G**AAAATCAA	811
	TTGATTTT**C**TTCCTTTT	812
Breast Cancer Gly-960-Asp GGC to GAC	ATAATGCCAAATGTAGTATCAAAGGAGGCTCTAGGTTTTGTCT ATCATCTCAGTTCAGAG**G**CAACGAAACTGGACTCATTACTCC AAATAAACATGGACTTTTACAAAACCCATATCGTAT	813

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATACGATATGGGTTTTGTAAAAGTCCATGTTTATTTGGAGTAA TGAGTCCAGTTTCGTTGCCTCTGAACTGAGATGATAGACAAA ACCTAGAGCCTCCTTTGATACTACATTTGGCATTAT	814
	GTTCAGAGGCAACGAAA	815
	TTTCGTTGCCTCTGAAC	816
Breast Cancer Met-1008-Ile ATG to ATA	ATTTGTTAAAACTAAATGTAAGAAAAATCTGCTAGAGGAAAAC TTTGAGGAACATTCAATGTCACCTGAAAGAGAAATGGGAAAT GAGAACATTCCAAGTACAGTGAGCACAATTAGCCGT	817
	ACGGCTAATTGTGCTCACTGTACTTGGAATGTTCTCATTTCCC ATTTCTCTTTCAGGTGACATTGAATGTTCCTCAAAGTTTTCCT CTAGCAGATTTTTCTTACATTTAGTTTTAACAAAT	818
	CATTCAATGTCACCTGA	819
	TCAGGTGACATTGAATG	820
Breast Cancer Thr-1025-Ile ACA to ATA	ACTTTGAGGAACATTCAATGTCACCTGAAAGAGAAATGGGAA ATGAGAACATTCCAAGTACAGTGAGCACAATTAGCCGTAATA ACATTAGAGAAAATGTTTTTAAAGAAGCCAGCTCAAG	821
	CTTGAGCTGGCTTCTTTAAAAACATTTTCTCTAATGTTATTACG GCTAATTGTGCTCACTGTACTTGGAATGTTCTCATTTCCCATT TCTCTTTCAGGTGACATTGAATGTTCCTCAAAGT	822
	TCCAAGTACAGTGAGCA	823
	TGCTCACTGTACTTGGA	824
Breast Cancer Glu-1038-Gly GAA to GGA	ACATTCCAAGTACAGTGAGCACAATTAGCCGTAATAACATTAG AGAAAATGTTTTTAAAGAAGCCAGCTCAAGCAATATTAATGAA GTAGGTTCCAGTACTAATGAAGTGGGCTCCAGTAT	825
	ATACTGGAGCCCACTTCATTAGTACTGGAACCTACTTCATTAA TATTGCTTGAGCTGGCTTCTTTAAAAACATTTTCTCTAATGTTA TTACGGCTAATTGTGCTCACTGTACTTGGAATGT	826
	TTTTAAAGAAGCCAGCT	827
	AGCTGGCTTCTTTAAAA	828
Breast Cancer Ser-1040-Asn AGC to AAC	CAAGTACAGTGAGCACAATTAGCCGTAATAACATTAGAGAAA ATGTTTTTAAAGAAGCCAGCTCAAGCAATATTAATGAAGTAGG TTCCAGTACTAATGAAGTGGGCTCCAGTATTAATGA	829
	TCATTAATACTGGAGCCCACTTCATTAGTACTGGAACCTACTT CATTAATATTGCTTGAGCTGGCTTCTTTAAAAACATTTTCTCTA ATGTTATTACGGCTAATTGTGCTCACTGTACTTG	830

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGAAGCCAGCTCAAGCA	831
	TGCTTGAGCTGGCTTCT	832
Breast Cancer Val-1047-Ala GTA to GCA	GCCGTAATAACATTAGAGAAAATGTTTTTAAAGAAGCCAGCTC AAGCAATATTAATGAAGTAGGTTCCAGTACTAATGAAGTGGG CTCCAGTATTAATGAAATAGGTTCCAGTGATGAAAA	833
	TTTTCATCACTGGAACCTATTTCATTAATACTGGAGCCCACTT CATTAGTACTGGAACCTACTTCATTAATATTGCTTGAGCTGGC TTCTTTAAAAACATTTTCTCTAATGTTATTACGGC	834
	TAATGAAGTAGGTTCCA	835
	TGGAACCTACTTCATTA	836
Breast Cancer Leu-1080-Stop TTG to TAG	AAATAGGTTCCAGTGATGAAAACATTCAAGCAGAACTAGGTA GAAACAGAGGGCCAAAATTGAATGCTATGCTTAGATTAGGGG TTTTGCAACCTGAGGTCTATAAACAAAGTCTTCCTGG	837
	CCAGGAAGACTTTGTTTATAGACCTCAGGTTGCAAAACCCCT AATCTAAGCATAGCATTCAAATTTTGGCCCTCTGTTTCTACCTA GTTCTGCTTGAATGTTTTCATCACTGGAACCTATTT	838
	GCCAAAATTGAATGCTA	839
	TAGCATTCAAATTTTGGC	840
Breast Cancer Leu-1086-Stop TTA to TGA	AAAACATTCAAGCAGAACTAGGTAGAAACAGAGGGCCAAAAT TGAATGCTATGCTTAGATTAGGGGTTTTGCAACCTGAGGTCT ATAAACAAAGTCTTCCTGGAAGTAATTGTAAGCATCC	841
	GGATGCTTACAATTACTTCCAGGAAGACTTTGTTTATAGACCT CAGGTTGCAAAACCCCTAATCTAAGCATAGCATTCAATTTTG GCCCTCTGTTTCTACCTAGTTCTGCTTGAATGTTTT	842
	GCTTAGATTAGGGGTTT	843
	AAACCCCTAATCTAAGC	844
Breast Cancer Ser-1130-Stop TCA to TGA	AGCAAGAATATGAAGAAGTAGTTCAGACTGTTAATACAGATTT CTCTCCATATCTGATTTCAGATAACTTAGAACAGCCTATGGGA AGTAGTCATGCATCTCAGGTTTGTTCTGAGACACC	845
	GGTGTCTCAGAACAAACCTGAGATGCATGACTACTTCCCATA GGCTGTTCTAAGTTATCTGAAATCAGATATGGAGAGAAATCT GTATTAACAGTCTGAACTACTTCTTCATATTCTTGCT	846
	TCTGATTTCAGATAACT	847
	AGTTATCTGAAATCAGA	848

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Lys-1183-Arg AAA to AGA	CTAGTTTTGCTGAAAATGACATTAAGGAAAGTTCTGCTGTTTT TAGCAAAAGCGTCCAGAAAGGAGAGCTTAGCAGGAGTCCTA GCCCTTTCACCCATACACATTTGGCTCAGGGTTACCG	849
	CGGTAACCCTGAGCCAAATGTGTATGGGTGAAAGGGCTAGG ACTCCTGCTAAGCTCTCCTTTCTGGACGCTTTTGCTAAAAACA GCAGAACTTTCCTTAATGTCATTTTCAGCAAAACTAG	850
	CGTCCAGAAAAGGAGAGC	851
	GCTCTCCTTTCTGGACG	852
Breast Cancer Gln-1200-Stop CAG to TAG	AGCGTCCAGAAAGGAGAGCTTAGCAGGAGTCCTAGCCCTTT CACCCATACACATTTGGCTCAGGGTTACCGAAGAGGGGCCA AGAAATTAGAGTCCTCAGAAGAGAACTTATCTAGTGAGG	853
	CCTCACTAGATAAGTTCTCTTCTGAGGACTCTAATTTCTTGGC CCCTCTTCGGTAACCCTGAGCCAAATGTGTATGGGTGAAAGG GCTAGGACTCCTGCTAAGCTCTCCTTTCTGGACGCT	854
	ATTTGGCTCAGGGTTAC	855
	GTAACCCTGAGCCAAAT	856
Breast Cancer Arg-1203-Stop CGA to TGA	AAAGGAGAGCTTAGCAGGAGTCCTAGCCCTTTCACCCATACA CATTTGGCTCAGGGTTACCGAAGAGGGGCCAAGAAATTAGA GTCCTCAGAAGAGAACTTATCTAGTGAGGATGAAGAGC	857
	GCTCTTCATCCTCACTAGATAAGTTCTCTTCTGAGGACTCTAA TTTCTTGGCCCCTCTTCGGTAACCCTGAGCCAAATGTGTATG GGTGAAAGGGCTAGGACTCCTGCTAAGCTCTCCTTT	858
	AGGGTTACCGAAGAGGG	859
	CCCTCTTCGGTAACCCT	860
Breast Cancer Glu-1214-Stop GAG to TAG	ACCCATACACATTTGGCTCAGGGTTACCGAAGAGGGGCCAA GAAATTAGAGTCCTCAGAAGAGAACTTATCTAGTGAGGATGA AGAGCTTCCCTGCTTCCAACACTTGTTATTTGGTAAAG	861
	CTTTACCAAATAACAAGTGTTGGAAGCAGGGAAGCTCTTCAT CCTCACTAGATAAGTTCTCTTCTGAGGACTCTAATTTCTTGGC CCCTCTTCGGTAACCCTGAGCCAAATGTGTATGGGT	862
	CCTCAGAAGAGAACTTA	863
	TAAGTTCTCTTCTGAGG	864
Breast Cancer Glu-1219-Asp GAG to GAC	TCAGGGTTACCGAAGAGGGGCCAAGAAATTAGAGTCCTCAG AAGAGAACTTATCTAGTGAGGATGAAGAGCTTCCCTGCTTCC AACACTTGTTATTTGGTAAAGTAAACAATATACCTTCT	865

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGAAGGTATATTGTTTACTTTACCAAATAACAAGTGTTGGAAG CAGGGAAGCTCTTCATCCTCACTAGATAAGTTCTCTTCTGAG GACTCTAATTTCTTGGCCCCTCTTCGGTAACCCTGA	866
	TCTAGTGAGGATGAAGA	867
	TCTTCATCCTCACTAGA	868
Breast Cancer Glu-1221-Stop GAA to TAA	GGTTACCGAAGAGGGGCCAAGAAATTAGAGTCCTCAGAAGA GAACTTATCTAGTGAGGATGAAGAGCTTCCCTGCTTCCAACA CTTGTTATTTGGTAAAGTAAACAATATACCTTCTCAGT	869
	ACTGAGAAGGTATATTGTTTACTTTACCAAATAACAAGTGTTG GAAGCAGGGAAGCTCTTCATCCTCACTAGATAAGTTCTCTTC TGAGGACTCTAATTTCTTGGCCCCTCTTCGGTAACC	870
	GTGAGGATGAAGAGCTT	871
	AAGCTCTTCATCCTCAC	872
Breast Cancer Glu-1250-Stop GAG to TAG	TTATTTGGTAAAGTAAACAATATACCTTCTCAGTCTACTAGGC ATAGCACCGTTGCTACCGAGTGTCTGTCTAAGAACACAGAGG AGAATTTATTATCATTGAAGAATAGCTTAAATGACT	873
	AGTCATTTAAGCTATTCTTCAATGATAATAAATTCTCCTCTGTG TTCTTAGACAGACACTCGGTAGCAACGGTGCTATGCCTAGTA GACTGAGAAGGTATATTGTTTACTTTACCAAATAA	874
	TTGCTACCGAGTGTCTG	875
	CAGACACTCGGTAGCAA	876
Breast Cancer Ser-1262-Stop TCA to TAA	CTAGGCATAGCACCGTTGCTACCGAGTGTCTGTCTAAGAACA CAGAGGAGAATTTATTATCATTGAAGAATAGCTTAAATGACTG CAGTAACCAGGTAATATTGGCAAAGGCATCTCAGGA	877
	TCCTGAGATGCCTTTGCCAATATTACCTGGTTACTGCAGTCAT TTAAGCTATTCTTCAATGATAATAAATTCTCCTCTGTGTTCTTA GACAGACACTCGGTAGCAACGGTGCTATGCCTAG	878
	TTTATTATCATTGAAGA	879
	TCTTCAATGATAATAAA	880
Breast Cancer Gln-1281-Stop CAG to TAG	TTATCATTGAAGAATAGCTTAAATGACTGCAGTAACCAGGTAA TATTGGCAAAGGCATCTCAGGAACATCACCTTAGTGAGGAAA CAAAATGTTCTGCTAGCTTGTTTTCTTCACAGTGCA	881
	TGCACTGTGAAGAAAACAAGCTAGCAGAACATTTTGTTTCCTC ACTAAGGTGATGTTCCTGAGATGCCTTTGCCAATATTACCTG GTTACTGCAGTCATTTAAGCTATTCTTCAATGATAA	882

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGGCATCTCAGGAACAT	883
	ATGTTCCTGAGATGCCT	884
Breast Cancer Gln-1313-Stop CAG to TAG	GCTAGCTTGTTTTCTTCACAGTGCAGTGAATTGGAAGACTTG ACTGCAAATACAAACACCCAGGATCCTTTCTTGATTGGTTCTT CCAAACAAATGAGGCATCAGTCTGAAAGCCAGGGAG	885
	CTCCCTGGCTTTCAGACTGATGCCTCATTTGTTTGGAAGAAC CAATCAAGAAAGGATCCTGGGTGTTTGTATTTGCAGTCAAGT CTTCCAATTCACTGCACTGTGAAGAAAACAAGCTAGC	886
	CAAACACCCAGGATCCT	887
	AGGATCCTGGGTGTTTG	888
Breast Cancer Ile-1318-Val ATT to GTT	TCACAGTGCAGTGAATTGGAAGACTTGACTGCAAATACAAAC ACCCAGGATCCTTTCTTGATTGGTTCTTCCAAACAAATGAGG CATCAGTCTGAAAGCCAGGGAGTTGGTCTGAGTGACA	889
	TGTCACTCAGACCAACTCCCTGGCTTTCAGACTGATGCCTCA TTTGTTTGGAAGAACCAATCAAGAAAGGATCCTGGGTGTTTG TATTTGCAGTCAAGTCTTCCAATTCACTGCACTGTGA	890
	CTTTCTTGATTGGTTCT	891
	AGAACCAATCAAGAAAG	892
Breast Cancer Gln-1323-Stop CAA to TAA	TTGGAAGACTTGACTGCAAATACAAACACCCAGGATCCTTTC TTGATTGGTTCTTCCAAACAAATGAGGCATCAGTCTGAAAGC CAGGGAGTTGGTCTGAGTGACAAGGAATTGGTTTCAG	893
	CTGAAACCAATTCCTTGTCACTCAGACCAACTCCCTGGCTTT CAGACTGATGCCTCATTTGTTTGGAAGAACCAATCAAGAAAG GATCCTGGGTGTTTGTATTTGCAGTCAAGTCTTCCAA	894
	CTTCCAAACAAATGAGG	895
	CCTCATTTGTTTGGAAG	896
Breast Cancer Arg-1347-Gly AGA to GGA	CAGTCTGAAAGCCAGGGAGTTGGTCTGAGTGACAAGGAATT GGTTTCAGATGATGAAGAAAGAGGAACGGGCTTGGAAGAAA ATAATCAAGAAGAGCAAAGCATGGATTCAAACTTAGGTA	897
	TACCTAAGTTTGAATCCATGCTTTGCTCTTCTTGATTATTTTCT TCCAAGCCCGTTCCTCTTTCTTCATCATCTGAAACCAATTCCT TGTCACTCAGACCAACTCCCTGGCTTTCAGACTG	898
	ATGAAGAAAGAGGAACG	899
	CGTTCCTCTTTCTTCAT	900

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Gln-1395-Stop CAG to TAG	GAAACAAGCGTCTCTGAAGACTGCTCAGGGCTATCCTCTCAG AGTGACATTTTAACCACTCAGGTAAAAAGCGTGTGTGTGTGT GCACATGCGTGTGTGTGGTGTCCTTTGCATTCAGTAG	901
	CTACTGAATGCAAAGGACACCACACACACGCATGTGCACACA CACACACGCTTTTTACCTGAGTGGTTAAAATGTCACTCTGAG AGGATAGCCCTGAGCAGTCTTCAGAGACGCTTGTTTC	902
	TAACCACTCAGGTAAAA	903
	TTTTACCTGAGTGGTTA	904
Breast Cancer Gln-1408-Stop CAG to TAG	TGGTGCCATTTATCGTTTTTGAAGCAGAGGGATACCATGCAA CATAACCTGATAAAGCTCCAGCAGGAAATGGCTGAACTAGAA GCTGTGTTAGAACAGCATGGGAGCCAGCCTTCTAACA	905
	TGTTAGAAGGCTGGCTCCCATGCTGTTCTAACACAGCTTCTA GTTCAGCCATTTCCTGCTGGAGCTTTATCAGGTTATGTTGCAT GGTATCCCTCTGCTTCAAAAACGATAAATGGCACCA	906
	TAAAGCTCCAGCAGGAA	907
	TTCCTGCTGGAGCTTTA	908
Breast Cancer Arg-1443-Gly CGA to GGA	AGCCAGCCTTCTAACAGCTACCCTTCCATCATAAGTGACTCT TCTGCCCTTGAGGACCTGCGAAATCCAGAACAAAGCACATCA GAAAAAGGTGTGTATTGTTGGCCAAACACTGATATCT	909
Arg-1443-Stop CGA to TGA	AGATATCAGTGTTTGGCCAACAATACACACCTTTTTCTGATGT GCTTTGTTCTGGATTTCGCAGGTCCTCAAGGGCAGAAGAGTC ACTTATGATGGAAGGGTAGCTGTTAGAAGGCTGGCT	910
	AGGACCTGCGAAATCCA	911
	TGGATTTCGCAGGTCCT	912
Breast Cancer Ser-1512-Ile AGT to ATT	CAGAATAGAAACTACCCATCTCAAGAGGAGCTCATTAAGGTT GTTGATGTGGAGGAGCAACAGCTGGAAGAGTCTGGGCCACA CGATTTGACGGAAACATCTTACTTGCCAAGGCAAGATC	913
	GATCTTGCCTTGGCAAGTAAGATGTTTCCGTCAAATCGTGTG GCCCAGACTCTTCCAGCTGTTGCTCCTCCACATCAACAACCT TAATGAGCTCCTCTTGAGATGGGTAGTTTCTATTCTG	914
	AGGAGCAACAGCTGGAA	915
	TTCCAGCTGTTGCTCCT	916
Breast Cancer Gln-1538-Stop CAG to TAG	ATCTTTCTAGGTCATCCCCTTCTAAATGCCCATCATTAGATGA TAGGTGGTACATGCACAGTTGCTCTGGGAGTCTTCAGAATAG AAACTACCCATCTCAAGAGGAGCTCATTAAGGTTGT	917

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACAACCTTAATGAGCTCCTCTTGAGATGGGTAGTTTCTATTCT GAAGACTCCCAGAGCAA**C**TGTGCATGTACCACCTATCATCTA ATGATGGGCATTTAGAAGGGGATGACCTAGAAAGAT	918
	CATGCACA**G**TTGCTCTG	919
	CAGAGCAA**C**TGTGCATG	920
Breast Cancer Glu-1541-Stop GAG to TAG	CAGAATAGAAACTACCCATCTCAAGAGGAGCTCATTAAGGTT GTTGATGTGGAGGAGCAA**C**AGCTGGAAGAGTCTGGGCCACA CGATTTGACGGAAACATCTTACTTGCCAAGGCAAGATC	921
	GATCTTGCCTTGGCAAGTAAGATGTTTCCGTCAAATCGTGTG GCCCAGACTCTTCCAGCT**G**TTGCTCCTCCACATCAACAACCT TAATGAGCTCCTCTTGAGATGGGTAGTTTCTATTCTG	922
	AGGAGCAA**C**AGCTGGAA	923
	TTCCAGCT**G**TTGCTCCT	924
Breast Cancer Thr-1561-Ile ACC to ATC	AACTACCCATCTCAAGAGGAGCTCATTAAGGTTGTTGATGTG GAGGAGCAACAGCTGGAA**G**AGTCTGGGCCACACGATTTGAC GGAAACATCTTACTTGCCAAGGCAAGATCTAGGTAATA	925
	TATTACCTAGATCTTGCCTTGGCAAGTAAGATGTTTCCGTCAA ATCGTGTGGCCCAGACT**C**TTCCAGCTGTTGCTCCTCCACATC AACAACCTTAATGAGCTCCTCTTGAGATGGGTAGTT	926
	AGCTGGAA**G**AGTCTGGG	927
	CCCAGACT**C**TTCCAGCT	928
Breast Cancer Tyr-1563-Stop TAC to TAG	TTTGTAATTCAACATTCATCGTTGTGTAAATTAAACTTCTCCCA TTCCTTTCAGAGGGAA**C**CCCTTACCTGGAATCTGGAATCAGC CTCTTCTCTGATGACCCTGAATCTGATCCTTCTGA	929
	TCAGAAGGATCAGATTCAGGGTCATCAGAGAAGAGGCTGATT CCAGATTCCAGGTAAGGG**G**TTCCCTCTGAAAGGAATGGGAG AAGTTTAATTTACACAACGATGAATGTTGAATTACAAA	930
	AGAGGGAA**C**CCCTTACC	931
	GGTAAGGG**G**TTCCCTCT	932
Breast Cancer Leu-1564-Pro CTG to CCG	CAACATTCATCGTTGTGTAAATTAAACTTCTCCCATTCCTTTC AGAGGGAACCCCTTACC**T**GGAATCTGGAATCAGCCTCTTCTC TGATGACCCTGAATCTGATCCTTCTGAAGACAGAGC	933
	GCTCTGTCTTCAGAAGGATCAGATTCAGGGTCATCAGAGAAG AGGCTGATTCCAGATTCC**A**GGTAAGGGGTTCCCTCTGAAAG GAATGGGAGAAGTTTAATTTACACAACGATGAATGTTG	934

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCCTTACCTGGAATCTG	935
	CAGATTCCAGGTAAGGG	936
Breast Cancer Gln-1604-Stop CAA to TAA	GCCCCAGAGTCAGCTCGTGTTGGCAACATACCATCTTCAACC TCTGCATTGAAAGTTCCCCAATTGAAAGTTGCAGAATCTGCC CAGAGTCCAGCTGCTGCTCATACTACTGATACTGCTG	937
	CAGCAGTATCAGTAGTATGAGCAGCAGCTGGACTCTGGGCA GATTCTGCAACTTTCAATTGGGGAACTTTCAATGCAGAGGTT GAAGATGGTATGTTGCCAACACGAGCTGACTCTGGGGC	938
	AAGTTCCCCAATTGAAA	939
	TTTCAATTGGGGAACTT	940
Breast Cancer Lys-1606-Glu AAA to GAA	GAGTCAGCTCGTGTTGGCAACATACCATCTTCAACCTCTGCA TTGAAAGTTCCCCAATTGAAAGTTGCAGAATCTGCCCAGAGT CCAGCTGCTGCTCATACTACTGATACTGCTGGGTATA	941
	TATACCCAGCAGTATCAGTAGTATGAGCAGCAGCTGGACTCT GGGCAGATTCTGCAACTTTCAATTGGGGAACTTTCAATGCAG AGGTTGAAGATGGTATGTTGCCAACACGAGCTGACTC	942
	CCCAATTGAAAGTTGCA	943
	TGCAACTTTCAATTGGG	944
Breast Cancer Met-1628-Thr ATG to ACG	CAGAATCTGCCCAGAGTCCAGCTGCTGCTCATACTACTGATA CTGCTGGGTATAATGCAATGGAAGAAAGTGTGAGCAGGGAG AAGCCAGAATTGACAGCTTCAACAGAAAGGGTCAACAA	945
	TTGTTGACCCTTTCTGTTGAAGCTGTCAATTCTGGCTTCTCCC TGCTCACACTTTCTTCCATTGCATTATACCCAGCAGTATCAGT AGTATGAGCAGCAGCTGGACTCTGGGCAGATTCTG	946
	TAATGCAATGGAAGAAA	947
	TTTCTTCCATTGCATTA	948
Breast Cancer Met-1628-Val ATG to GTG	GCAGAATCTGCCCAGAGTCCAGCTGCTGCTCATACTACTGAT ACTGCTGGGTATAATGCAATGGAAGAAAGTGTGAGCAGGGA GAAGCCAGAATTGACAGCTTCAACAGAAAGGGTCAACA	949
	TGTTGACCCTTTCTGTTGAAGCTGTCAATTCTGGCTTCTCCCT GCTCACACTTTCTTCCATTGCATTATACCCAGCAGTATCAGTA GTATGAGCAGCAGCTGGACTCTGGGCAGATTCTGC	950
	ATAATGCAATGGAAGAA	951

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTCTTCCATTGCATTAT	952
Breast Cancer Pro-1637-Leu CCA to CTA	CTCATACTACTGATACTGCTGGGTATAATGCAATGGAAGAAA GTGTGAGCAGGGAGAAGC**C**AGAATTGACAGCTTCAACAGAA AGGGTCAACAAAAGAATGTCCATGGTGGTGTCTGGCCT	953
	AGGCCAGACACCACCATGGACATTCTTTTGTTGACCCTTTCT GTTGAAGCTGTCAATTCT**G**GCTTCTCCCTGCTCACACTTTCTT CCATTGCATTATACCCAGCAGTATCAGTAGTATGAG	954
	GGAGAAGC**C**AGAATTGA	955
	TCAATTCT**G**GCTTCTCC	956
Breast Cancer Met-1652-Ile ATG to ATA	GAGCAGGGAGAAGCCAGAATTGACAGCTTCAACAGAAAGGG TCAACAAAAGAATGTCCAT**G**GTGGTGTCTGGCCTGACCCCAG AAGAATTTGTGAGTGTATCCATATGTATCTCCCTAATG	957
	CATTAGGGAGATACATATGGATACACTCACAAATTCTTCTGG GGTCAGGCCAGACACCAC**C**ATGGACATTCTTTTGTTGACCCT TTCTGTTGAAGCTGTCAATTCTGGCTTCTCCCTGCTC	958
	ATGTCCAT**G**GTGGTGTC	959
	GACACCAC**C**ATGGACAT	960
Breast Cancer Glu-1694-Stop GAG to TAG	CACTTCCTGATTTTGTTTTCAACTTCTAATCCTTTGAGTGTTTT TCATTCTGCAGATGCT**G**AGTTTGTGTGTGAACGGACACTGAA ATATTTTCTAGGAATTGCGGGAGGAAAATGGGTAG	961
	CTACCCATTTTCCTCCCGCAATTCCTAGAAAATATTTCAGTGT CCGTTCACACACAAACT**C**AGCATCTGCAGAATGAAAAACACT CAAAGGATTAGAAGTTGAAAACAAAATCAGGAAGTG	962
	CAGATGCT**G**AGTTTGTG	963
	CACAAACT**C**AGCATCTG	964
Breast Cancer Gly-1706-Glu GGA to GAA	GTGTTTTTCATTCTGCAGATGCTGAGTTTGTGTGTGAACGGA CACTGAAATATTTTCTAG**G**AATTGCGGGAGGAAAATGGGTAG TTAGCTATTTCTGTAAGTATAATACTATTTCTCCCCT	965
	AGGGGAGAAATAGTATTATACTTACAGAAATAGCTAACTACCC ATTTTCCTCCCGCAATT**C**CTAGAAAATATTTCAGTGTCCGTTC ACACACAAACTCAGCATCTGCAGAATGAAAAACAC	966
	TTTTCTAG**G**AATTGCGG	967
	CCGCAATT**C**CTAGAAAA	968

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Ala-1708-Glu GCG to GAG	TTCATTCTGCAGATGCTGAGTTTGTGTGTGAACGGACACTGA AATATTTTCTAGGAATTG**C**GGGAGGAAAATGGGTAGTTAGCT ATTTCTGTAAGTATAATACTATTTCTCCCCTCCTCCC	969
	GGGAGGAGGGGAGAAATAGTATTATACTTACAGAAATAGCTA ACTACCCATTTTCCTCCC**G**CAATTCCTAGAAAATATTTCAGTG TCCGTTCACACACAAACTCAGCATCTGCAGAATGAA	970
	AGGAATTG**C**GGGAGGAA	971
	TTCCTCCC**G**CAATTCCT	972
Breast Cancer Val-1713-Ala GTA to GCA	CTGAGTTTGTGTGTGAACGGACACTGAAATATTTTCTAGGAAT TGCGGGAGGAAAATGGG**T**AGTTAGCTATTTCTGTAAGTATAA TACTATTTCTCCCCTCCTCCCTTTAACACCTCAGAA	973
	TTCTGAGGTGTTAAAGGGAGGAGGGGAGAAATAGTATTATAC TTACAGAAATAGCTAACT**A**CCCATTTTCCTCCCGCAATTCCTA GAAAATATTTCAGTGTCCGTTCACACACAAACTCAG	974
	AAAATGGG**T**AGTTAGCT	975
	AGCTAACT**A**CCCATTTT	976
Breast Cancer Trp-1718-Stop TGG to TAG	AACGGACACTGAAATATTTTCTAGGAATTGCGGGAGGAAAAT GGGTAGTTAGCTATTTCT**G**TAAGTATAATACTATTTCTCCCCT CCTCCCTTTAACACCTCAGAATTGCATTTTTACACC	977
	GGTGTAAAAATGCAATTCTGAGGTGTTAAAGGGAGGAGGGG AGAAATAGTATTATACTTA**C**AGAAATAGCTAACTACCCATTTTC CTCCCGCAATTCCTAGAAAATATTTCAGTGTCCGTT	978
	CTATTTCT**G**TAAGTATA	979
	TATACTTA**C**AGAAATAG	980
Breast Cancer Glu-1725-Stop GAA to TAA	TTCTGCTGTATGTAACCTGTCTTTTCTATGATCTCTTTAGGGG TGACCCAGTCTATTAAA**G**AAAGAAAAATGCTGAATGAGGTAA GTACTTGATGTTACAAACTAACCAGAGATATTCATT	981
	AATGAATATCTCTGGTTAGTTTGTAACATCAAGTACTTACCTC ATTCAGCATTTTTCTTT**C**TTTAATAGACTGGGTCACCCCTAAA GAGATCATAGAAAAGACAGGTTACATACAGCAGAA	982
	CTATTAAA**G**AAAGAAAA	983
	TTTTCTTT**C**TTTAATAG	984
Breast Cancer Lys-1727-Stop AAA to TAA	TGTATGTAACCTGTCTTTTCTATGATCTCTTTAGGGGTGACCC AGTCTATTAAAGAAAGA**A**AAATGCTGAATGAGGTAAGTACTTG ATGTTACAAACTAACCAGAGATATTCATTCAGTCA	985

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGACTGAATGAATATCTCTGGTTAGTTTGTAACATCAAGTACT TACCTCATTCAGCATTTTTCTTTCTTTAATAGACTGGGTCACC CCTAAAGAGATCATAGAAAAGACAGGTTACATACA	986
	AAGAAAGAAAAATGCTG	987
	CAGCATTTTTCTTTCTT	988
Breast Cancer Pro-1749-Arg CCA to CGA	TCTTTCAGCATGATTTTGAAGTCAGAGGAGATGTGGTCAATG GAAGAAACCACCAAGGTCCAAAGCGAGCAAGAGAATCCCAG GACAGAAAGGTAAAGCTCCCTCCCTCAAGTTGACAAAA	989
	TTTTGTCAACTTGAGGGAGGGAGCTTTACCTTTCTGTCCTGG GATTCTCTTGCTCGCTTTGGACCTTGGTGGTTTCTTCCATTGA CCACATCTCCTCTGACTTCAAAATCATGCTGAAAGA	990
	CCAAGGTCCAAAGCGAG	991
	CTCGCTTTGGACCTTGG	992
Breast Cancer Arg-1751-Stop CGA to TGA	CAGCATGATTTTGAAGTCAGAGGAGATGTGGTCAATGGAAGA AACCACCAAGGTCCAAAGCGAGCAAGAGAATCCCAGGACAG AAAGGTAAAGCTCCCTCCCTCAAGTTGACAAAAATCTC	993
	GAGATTTTTGTCAACTTGAGGGAGGGAGCTTTACCTTTCTGT CCTGGGATTCTCTTGCTCGCTTTGGACCTTGGTGGTTTCTTC CATTGACCACATCTCCTCTGACTTCAAAATCATGCTG	994
	GTCCAAAGCGAGCAAGA	995
	TCTTGCTCGCTTTGGAC	996
Breast Cancer Gln-1756-Stop CAG to TAG	GTCAGAGGAGATGTGGTCAATGGAAGAAACCACCAAGGTCC AAAGCGAGCAAGAGAATCCCAGGACAGAAAGGTAAAGCTCC CTCCCTCAAGTTGACAAAAATCTCACCCCACCACTCTGT	997
	ACAGAGTGGTGGGGTGAGATTTTTGTCAACTTGAGGGAGGG AGCTTTACCTTTCTGTCCTGGGATTCTCTTGCTCGCTTTGGA CCTTGGTGGTTTCTTCCATTGACCACATCTCCTCTGAC	998
	GAGAATCCCAGGACAGA	999
	TCTGTCCTGGGATTCTC	1000
Breast Cancer Met-1775-Arg ATG to AGG	CTCTCTTCTTCCAGATCTTCAGGGGGCTAGAAATCTGTTGCT ATGGGCCCTTCACCAACATGCCCACAGGTAAGAGCCTGGGA GAACCCCAGAGTTCCAGCACCAGCCTTTGTCTTACATA	1001
	TATGTAAGACAAAGGCTGGTGCTGGAACTCTGGGGTTCTCCC AGGCTCTTACCTGTGGGCATGTTGGTGAAGGGCCCATAGCA ACAGATTTCTAGCCCCCTGAAGATCTGGAAGAAGAGAG	1002

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CACCAACATGCCCACAG	1003
	CTGTGGGCATGTTGGTG	1004
Breast Cancer Trp-1782-Stop TGG to TGA	AGTATGCAGATTACTGCAGTGATTTTACATCTAAATGTCCATT TTAGATCAACTGGAATGGATGGTACAGCTGTGTGGTGCTTCT GTGGTGAAGGAGCTTTCATCATTCACCCTTGGCACA	1005
	TGTGCCAAGGGTGAATGATGAAAGCTCCTTCACCACAGAAGC ACCACACAGCTGTACCATCCATTCCAGTTGATCTAAAATGGA CATTTAGATGTAAAATCACTGCAGTAATCTGCATACT	1006
	CTGGAATGGATGGTACA	1007
	TGTACCATCCATTCCAG	1008
Breast Cancer Gln-1785-His CAG to CAT	ATTACTGCAGTGATTTTACATCTAAATGTCCATTTTAGATCAAC TGGAATGGATGGTACAGCTGTGTGGTGCTTCTGTGGTGAAG GAGCTTTCATCATTCACCCTTGGCACAGTAAGTATT	1009
	AATACTTACTGTGCCAAGGGTGAATGATGAAAGCTCCTTCAC CACAGAAGCACCACACAGCTGTACCATCCATTCCAGTTGATC TAAAATGGACATTTAGATGTAAAATCACTGCAGTAAT	1010
	ATGGTACAGCTGTGTGG	1011
	CCACACAGCTGTACCAT	1012
Breast Cancer Glu-1794-Asp GAG to GAT	GTCCATTTTAGATCAACTGGAATGGATGGTACAGCTGTGTGG TGCTTCTGTGGTGAAGGAGCTTTCATCATTCACCCTTGGCAC AGTAAGTATTGGGTGCCCTGTCAGAGAGGGAGGACAC	1013
	GTGTCCTCCCTCTCTGACAGGGCACCCAATACTTACTGTGCC AAGGGTGAATGATGAAAGCTCCTTCACCACAGAAGCACCACA CAGCTGTACCATCCATTCCAGTTGATCTAAAATGGAC	1014
	GTGAAGGAGCTTTCATC	1015
	GATGAAAGCTCCTTCAC	1016
Breast Cancer Arg-1835-Stop CGA to TGA	CTCTGCTTGTGTTCTCTGTCTCCAGCAATTGGGCAGATGTGT GAGGCACCTGTGGTGACCCGAGAGTGGGTGTTGGACAGTGT AGCACTCTACCAGTGCCAGGAGCTGGACACCTACCTGA	1017
	TCAGGTAGGTGTCCAGCTCCTGGCACTGGTAGAGTGCTACA CTGTCCAACACCCACTCTCGGGTCACCACAGGTGCCTCACA CATCTGCCCAATTGCTGGAGACAGAGAACACAAGCAGAG	1018
	TGGTGACCCGAGAGTGG	1019
	CCACTCTCGGGTCACCA	1020

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Trp-1837-Arg TGG to CGG	TTGTGTTCTCTGTCTCCAGCAATTGGGCAGATGTGTGAGGCA CCTGTGGTGACCCGAGAGTGGGTGTTGGACAGTGTAGCACT CTACCAGTGCCAGGAGCTGGACACCTACCTGATACCCC	1021
	GGGGTATCAGGTAGGTGTCCAGCTCCTGGCACTGGTAGAGT GCTACACTGTCCAACACCCACTCTCGGGTCACCACAGGTGC CTCACACATCTGCCCAATTGCTGGAGACAGAGAACACAA	1022
	CCCGAGAGTGGGTGTTG	1023
	CAACACCCACTCTCGGG	1024
Breast Cancer Trp-1837-Stop TGG to TAG	TGTGTTCTCTGTCTCCAGCAATTGGGCAGATGTGTGAGGCAC CTGTGGTGACCCGAGAGTGGGTGTTGGACAGTGTAGCACTC TACCAGTGCCAGGAGCTGGACACCTACCTGATACCCCA	1025
	TGGGGTATCAGGTAGGTGTCCAGCTCCTGGCACTGGTAGAG TGCTACACTGTCCAACACCCACTCTCGGGTCACCACAGGTG CCTCACACATCTGCCCAATTGCTGGAGACAGAGAACACA	1026
	CCGAGAGTGGGTGTTGG	1027
	CCAACACCCACTCTCGG	1028

Table 15
BRCA2 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast cancer PHE32LEU TTT to CTT	GTTAAAACTAAGGTGGGATTTTTTTTTTAAATAGATTTAGGAC CAATAAGTCTTAATTGGTTTGAAGAACTTTCTTCAGAAGCTCC ACCCTATAATTCTGAACCTGCAGAAGAATCTGAAC	1029
	GTTCAGATTCTTCTGCAGGTTCAGAATTATAGGGTGGAGCTT CTGAAGAAAGTTCTTCAAACCAATTAAGACTTATTGGTCCTAA ATCTATTTAAAAAAAAAATCCCACCTTAGTTTTAAC	1030
	TTAATTGGTTTGAAGAA	1031
	TTCTTCAAACCAATTAA	1032
Breast cancer TYR42CYS TAT to TGT	TAGATTTAGGACCAATAAGTCTTAATTGGTTTGAAGAACTTTC TTCAGAAGCTCCACCCTATAATTCTGAACCTGCAGAAGAATC TGAACATAAAAACAACAATTACGAACCAAACCTATT	1033

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AATAGGTTTGGTTCGTAATTGTTGTTTTTATGTTCAGATTCTTC TGCAGGTTCAGAATTATAGGGTGGAGCTTCTGAAGAAAGTTC TTCAAACCAATTAAGACTTATTGGTCCTAAATCTA	1034
	TCCACCCTATAATTCTG	1035
	CAGAATTATAGGGTGGA	1036
Breast cancer LYS53ARG AAA to AGA	AAGAACTTTCTTCAGAAGCTCCACCCTATAATTCTGAACCTGC AGAAGAATCTGAACATAAAAACAACAATTACGAACCAAACCTA TTTAAAACTCCACAAAGGAAACCATCTTATAATCA	1037
	TGATTATAAGATGGTTTCCTTTGTGGAGTTTTAAATAGGTTTG GTTCGTAATTGTTGTTTTTATGTTCAGATTCTTCTGCAGGTTC AGAATTATAGGGTGGAGCTTCTGAAGAAAGTTCTT	1038
	TGAACATAAAAACAACA	1039
	TGTTGTTTTTATGTTCA	1040
Breast cancer Phe81Leu TTC to CTC	CTATTTAAAACTCCACAAAGGAAACCATCTTATAATCAGCTGG CTTCAACTCCAATAATATTTCAAAGAGCAAGGGCTGACTCTGC CGCTGTACCAATCTCCTGTAAAAGAATTAGATAAAT	1041
	ATTTATCTAATTCTTTTACAGGAGATTGGTACAGCGGCAGAGT CAGCCCTTGCTCTTTGAATATTATTGGAGTTGAAGCCAGCTG ATTATAAGATGGTTTCCTTTGTGGAGTTTTAAATAG	1042
	CAATAATATTCAAAGAG	1043
	CTCTTTGAATATTATTG	1044
Breast cancer TRP194TERM TGG to TAG	GTCAGACACCAAAACATATTTCTGAAAGTCTAGGAGCTGAGG TGGATCCTGATATGTCTTGGTCAAGTTCTTTAGCTACACCACC CACCCTTAGTTCTACTGTGCTCATAGGTAATAATAG	1045
	CTATTATTACCTATGAGCACAGTAGAACTAAGGGTGGGTGGT GTAGCTAAAGAACTTGACCAAGACATATCAGGATCCACCTCA GCTCCTAGACTTTCAGAAATATGTTTTGGTGTCTGAC	1046
	TATGTCTTGGTCAAGTT	1047
	AACTTGACCAAGACATA	1048
Breast cancer PRO201ARG CCA to CGA	CTGAAAGTCTAGGAGCTGAGGTGGATCCTGATATGTCTTGGT CAAGTTCTTTAGCTACACCACCCACCCTTAGTTCTACTGTGCT CATAGGTAATAATAGCAAATGTGTATTTACAAGAAA	1049
	TTTCTTGTAAATACACATTTGCTATTATTACCTATGAGCACAGT AGAACTAAGGGTGGGTGGTGTAGCTAAAGAACTTGACCAAGA CATATCAGGATCCACCTCAGCTCCTAGACTTTCAG	1050

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGCTACACCACCCACCC	1051
	GGGTGGGTGGTGTAGCT	1052
Breast cancer Pro222Ser CCT to TCT	ACAATACACATAAATTTTTATCTTACAGTCAGAAATGAAGAAG CATCTGAAACTGTATTTCCTCATGATACTACTGCTGTAAGTAA ATATGACATTGATTAGACTGTTGAAATTGCTAACA	1053
	TGTTAGCAATTTCAACAGTCTAATCAATGTCATATTTACTTACA GCAGTAGTATCATGAGGAAATACAGTTTCAGATGCTTCTTCAT TTCTGACTGTAAGATAAAAATTTATGTGTATTGT	1054
	CTGTATTTCCTCATGAT	1055
	ATCATGAGGAAATACAG	1056
Breast cancer Leu-414-Term TTG to TAG	AATGGTCTCAACTAACCCTTTCAGGTCTAAATGGAGCCCAGA TGGAGAAAATACCCCTATTGCATATTTCTTCATGTGACCAAAA TATTTCAGAAAAAGACCTATTAGACACAGAGAACAA	1057
	TTGTTCTCTGTGTCTAATAGGTCTTTTTCTGAAATATTTTGGTC ACATGAAGAAATATGCAATAGGGGTATTTTCTCCATCTGGGC TCCATTTAGACCTGAAAGGGTTAGTTGAGACCATT	1058
	ACCCCTATTGCATATTT	1059
	AAATATGCAATAGGGGT	1060
Breast cancer, male Cys554Trp TGT to TGG	AGCCTCTGAAAGTGGACTGGAAATACATACTGTTTGCTCACA GAAGGAGGACTCCTTATGTCCAAATTTAATTGATAATGGAAG CTGGCCAGCCACCACCACACAGAATTCTGTAGCTTTG	1061
	CAAAGCTACAGAATTCTGTGTGGTGGTGGCTGGCCAGCTTC CATTATCAATTAAATTTGGACATAAGGAGTCCTCCTTCTGTGA GCAAACAGTATGTATTTCCAGTCCACTTTCAGAGGCT	1062
	TCCTTATGTCCAAATTT	1063
	AAATTTGGACATAAGGA	1064
Breast cancer Lys944Term AAA to TAA	AACTCTACCATGGTTTTATATGGAGACACAGGTGATAAACAA GCAACCCAAGTGTCAATTAAAAAAGATTTGGTTTATGTTCTTG CAGAGGAGAACAAAAATAGTGTAAAGCAGCATATAA	1065
	TTATATGCTGCTTTACACTATTTTTGTTCTCCTCTGCAAGAAC ATAAACCAAATCTTTTTTAATTGACACTTGGGTTGCTTGTTTAT CACCTGTGTCTCCATATAAAACCATGGTAGAGTT	1066
	TGTCAATTAAAAAAGAT	1067
	ATCTTTTTTAATTGACA	1068

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast cancer, male Glu1320Term GAA to TAA	ATGACTACTGGCACTTTTGTTGAAGAAATTACTGAAAATTACA AGAGAAATACTGAAAAT**G**AAGATAACAAATATACTGCTGCCAG TAGAAATTCTCATAACTTAGAATTTGATGGCAGTG	1069
	CACTGCCATCAAATTCTAAGTTATGAGAATTTCTACTGGCAGC AGTATATTTGTTATCTT**C**ATTTTCAGTATTTCTCTTGTAATTTTC AGTAATTTCTTCAACAAAAGTGCCAGTAGTCAT	1070
	CTGAAAAT**G**AAGATAAC	1071
	GTTATCTT**C**ATTTTCAG	1072
Breast cancer Glu1876Term GAA to TAA	CATGAAACAATTAAAAAAGTGAAAGACATATTTACAGACAGTT TCAGTAAAGTAATTAAG**G**AAAACAACGAGAATAAATCAAAAAT TTGCCAAACGAAAATTATGGCAGGTTGTTACGAGG	1073
	CCTCGTAACAACCTGCCATAATTTTCGTTTGGCAAATTTTTGA TTTATTCTCGTTGTTTT**C**CTTAATTACTTTACTGAAACTGTCTG TAAATATGTCTTTCACTTTTTTAATTGTTTCATG	1074
	TAATTAAG**G**AAAACAAC	1075
	GTTGTTTT**C**CTTAATTA	1076
Breast cancer Ser1882Term TCA to TAA	TGAAAGACATATTTACAGACAGTTTCAGTAAAGTAATTAAGGA AAACAACGAGAATAAAT**C**AAAAATTTGCCAAACGAAAATTATG GCAGGTTGTTACGAGGCATTGGATGATTCAGAGGA	1077
	TCCTCTGAATCATCCAATGCCTCGTAACAACCTGCCATAATTT TCGTTTGGCAAATTTTT**G**ATTTATTCTCGTTGTTTTCCTTAATT ACTTTACTGAAACTGTCTGTAAATATGTCTTTCA	1078
	GAATAAAT**C**AAAAATTT	1079
	AAATTTTT**G**ATTTATTC	1080
Breast cancer Glu1953Term GAA to TAA	AACCAAAATATGTCTGGATTGGAGAAAGTTTCTAAAATATCAC CTTGTGATGTTAGTTTG**G**AAACTTCAGATATATGTAAATGTAG TATAGGGAAGCTTCATAAGTCAGTCTCATCTGCAA	1081
	TTGCAGATGAGACTGACTTATGAAGCTTCCCTATACTACATTT ACATATATCTGAAGTTT**C**CAAACTAACATCACAAGGTGATATT TTAGAAACTTTCTCCAATCCAGACATATTTTGGTT	1082
	TTAGTTTG**G**AAACTTCA	1083
	TGAAGTTT**C**CAAACTAA	1084
Breast cancer Ser1970Term TCA to TAA	TTAGTTTGGAAACTTCAGATATATGTAAATGTAGTATAGGGAA GCTTCATAAGTCAGTCT**C**ATCTGCAAATACTTGTGGGATTTTT AGCACAGCAAGTGGAAAATCTGTCCAGGTATCAGA	1085

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCTGATACCTGGACAGATTTTCCACTTGCTGTGCTAAAAATCC CACAAGTATTTGCAGATGAGACTGACTTATGAAGCTTCCCTAT ACTACATTTACATATATCTGAAGTTTCCAAACTAA	1086
	GTCAGTCTCATCTGCAA	1087
	TTGCAGATGAGACTGAC	1088
Breast cancer Gln1987Term CAG to TAG	AAGTCAGTCTCATCTGCAAATACTTGTGGGATTTTTAGCACAG CAAGTGGAAAATCTGTCCAGGTATCAGATGCTTCATTACAAAA CGCAAGACAAGTGTTTTCTGAAATAGAAGATAGTA	1089
	TACTATCTTCTATTTCAGAAAACACTTGTCTTGCGTTTTGTAAT GAAGCATCTGATACCTGGACAGATTTTCCACTTGCTGTGCTA AAAATCCCACAAGTATTTGCAGATGAGACTGACTT	1090
	AATCTGTCCAGGTATCA	1091
	TGATACCTGGACAGATT	1092
Breast cancer Ala2466Val GCA to GTA	AAAATAAGATTAATGACAATGAGATTCATCAGTTTAACAAAAA CAACTCCAATCAAGCAGCAGCTGTAACTTTCACAAAGTGTGA AGAAGAACCTTTAGGTATTGTATGACAATTTGTGTG	1093
	CACACAAATTGTCATACAATACCTAAAGGTTCTTCTTCACACT TTGTGAAAGTTACAGCTGCTGCTTGATTGGAGTTGTTTTTGTT AAACTGATGAATCTCATTGTCATTAATCTTATTTT	1094
	TCAAGCAGCAGCTGTAA	1095
	TTACAGCTGCTGCTTGA	1096
Breast cancer Arg2520Term CGA to TGA	AGGCAACGCGTCTTTCCACAGCCAGGCAGTCTGTATCTTGCA AAAACATCCACTCTGCCTCGAATCTCTCTGAAAGCAGCAGTA GGAGGCCAAGTCCCCTCTGCGTGTCCTCATAAACAGG	1097
	CCTGTTTATGAGGACACGCAGAGGGGACTTGGCCTCCTACT GCTGCTTTCAGAGAGATTCGAGGCAGAGTGGATGTTTTTGCA AGATACAGACTGCCTGGCTGTGGAAAGACGCGTTGCCT	1098
	CTCTGCCTCGAATCTCT	1099
	AGAGATTCGAGGCAGAG	1100
Breast cancer Gln2714Term CAA to TAA	ATTTCATTGAGCGCAAATATATCTGAAACTTCTAGCAATAAAA CTAGTAGTGCAGATACCCAAAAAGTGGCCATTATTGAACTTA CAGATGGGTGGTATGCTGTTAAGGCCCAGTTAGATC	1101
	GATCTAACTGGGCCTTAACAGCATACCACCCATCTGTAAGTT CAATAATGGCCACTTTTTGGGTATCTGCACTACTAGTTTTATT GCTAGAAGTTTCAGATATATTTGCGCTCAATGAAAT	1102

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CAGATACCCAAAAAGTG	1103
	CACTTTTTGGGTATCTG	1104
Breast cancer Leu2776Term TTA to TGA	CAGAACTGGTGGGCTCTCCTGATGCCTGTACACCTCTTGAAG CCCCAGAATCTCTTATGTTAAAGGTAAATTAATTTGCACTCTT GGTAAAAATCAGTCATTGATTCAGTTAAATTCTAGA	1105
	TCTAGAATTTAACTGAATCAATGACTGATTTTTACCAAGAGTG CAAATTAATTTACCTTTAACATAAGAGATTCTGGGGCTTCAAG AGGTGTACAGGCATCAGGAGAGCCCACCAGTTCTG	1106
	TCTTATGTTAAAGATTT	1107
	AAATCTTTAACATAAGA	1108
Breast cancer Gln2893Term CAG to TAG	CCTTTTGTTTTCTTAGAAAACACAACAAAACCATATTTACCATC ACGTGCACTAACAAGACAGCAAGTTCGTGCTTTGCAAGATGG TGCAGAGCTTTATGAAGCAGTGAAGAATGCAGCAG	1109
	CTGCTGCATTCTTCACTGCTTCATAAAGCTCTGCACCATCTTG CAAAGCACGAACTTGCTGTCTTGTTAGTGCACGTGATGGTAA ATATGGTTTTGTTGTGTTTTCTAAGAAAACAAAAGG	1110
	TAACAAGACAGCAAGTT	1111
	AACTTGCTGTCTTGTTA	1112
Breast cancer Ala2951Thr GCC to ACC	AATCACAGGCAAATGTTGAATGATAAGAAACAAGCTCAGATC CAGTTGGAAATTAGGAAGGCCATGGAATCTGCTGAACAAAAG GAACAAGGTTTATCAAGGGATGTCACAACCGTGTGGA	1113
	TCCACACGGTTGTGACATCCCTTGATAAACCTTGTTCCTTTTG TTCAGCAGATTCCATGGCCTTCCTAATTTCCAACTGGATCTGA GCTTGTTTCTTATCATTCAACATTTGCCTGTGATT	1114
	TTAGGAAGGCCATGGAA	1115
	TTCCATGGCCTTCCTAA	1116
Breast cancer Met3118Thr ATG to ACG	ACAATTTACTGGCAATAAAGTTTTGGATAGACCTTAATGAGGA CATTATTAAGCCTCATATGTTAATTGCTGCAAGCAACCTCCAG TGGCGACCAGAATCCAAATCAGGCCTTCTTACTTT	1117
	AAAGTAAGAAGGCCTGATTTGGATTCTGGTCGCCACTGGAG GTTGCTTGCAGCAATTAACATATGAGGCTTAATAATGTCCTCA TTAAGGTCTATCCAAAACTTTATTGCCAGTAAATTGT	1118
	GCCTCATATGTTAATTG	1119
	CAATTAACATATGAGGC	1120

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast cancer Thr3401Met ACG to ATG	GACTGAAACGACGTTGTACTACATCTCTGATCAAAGAACAGG AGAGTTCCCAGGCCAGTA**C**GGAAGAATGTGAGAAAAATAAGC AGGACACAATTACAACTAAAAAATATATCTAAGCATT	1121
	AATGCTTAGATATATTTTTTAGTTGTAATTGTGTCCTGCTTATT TTTCTCACATTCTTCC**G**TACTGGCCTGGGAACTCTCCTGTTCT TTGATCAGAGATGTAGTACAACGTCGTTTCAGTC	1122
	GGCCAGTA**C**GGAAGAAT	1123
	ATTCTTCC**G**TACTGGCC	1124
Breast cancer Ile3412Val ATT to GTT	AAAGAACAGGAGAGTTCCCAGGCCAGTACGGAAGAATGTGA GAAAAATAAGCAGGACACA**A**TTACAACTAAAAAATATATCTAA GCATTTGCAAAGGCGACAATAAATTATTGACGCTTAA	1125
	TTAAGCGTCAATAATTTATTGTCGCCTTTGCAAATGCTTAGAT ATATTTTTTAGTTGTAA**T**TGTGTCCTGCTTATTTTTCTCACATT CTTCCGTACTGGCCTGGGAACTCTCCTGTTCTTT	1126
	AGGACACA**A**TTACAACT	1127
	AGTTGTAA**T**TGTGTCCT	1128

EXAMPLE 9
Cystic Fibrosis - CFTR

Cystic fibrosis is a lethal disease affecting approximately one in 2,500 live Caucasian births and is the most common autosomal recessive disease in Caucasians. Patients with this disease have reduced chloride ion permeability in the secretory and absorptive cells of organs with epithelial cell linings, including the airways, pancreas, intestine, sweat glands and male genital tract. This, in turn, reduces the transport of water across the epithelia. The lungs and the GI tract are the predominant organ systems affected in this disease and the pathology is characterized by blocking of the respiratory and GI tracts with viscous mucus. The chloride impermeability in affected tissues is due to mutations in a specific chloride channel, the cystic fibrosis transmembrane conductance regulator protein (CFTR), which prevents normal passage of chloride ions through the cell membrane (Welsh et al., Neuron, 8:821-829 (1992)). Damage to the lungs due to mucus blockage, frequent bacterial infections and inflammation is the primary cause of morbidity and mortality in CF patients and, although maintenance therapy has improved the quality of patients' lives, the median age at death is still only around 30 years. There is no effective treatment for the disease, and therapeutic research is focused on gene therapy using

exogenous transgenes in viral vectors and/or activating the defective or other chloride channels in the cell membrane to normalize chloride permeability (Tizzano et al., J. Pediat., 120:337-349 (1992)). However, the death of a teenage patient treated with an adenovirus vector carrying an exogenous CFTR gene in clinical trials in the late 1990's has impacted this area of research.

The oligonucleotides of the invention for correction of the CFTR gene are attached as a table.

Table 16
CFTR Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Ala46Asp GCT to GAT	AAGGATACAGACAGCGCCTGGAATTGTCAGACATATACCAAA TCCCTTCTGTTGATTCTG**C**TGACAATCTATCTGAAAAATTGGA AAGGTATGTTCATGTACATTGTTTAGTTGAAGAGAG	1129
	CTCTCTTCAACTAAACAATGTACATGAACATACCTTTCCAATTT TTCAGATAGATTGTCA**G**CAGAATCAACAGAAGGGATTTGGTA TATGTCTGACAATTCCAGGCGCTGTCTGTATCCTT	1130
	TGATTCTG**C**TGACAATC	1131
	GATTGTCA**G**CAGAATCA	1132
Cystic fibrosis Ser50Tyr TCT to TAT	AGCGCCTGGAATTGTCAGACATATACCAAATCCCTTCTGTTG ATTCTGCTGACAATCTAT**C**TGAAAAATTGGAAAGGTATGTTCA TGTACATTGTTTAGTTGAAGAGAGAAATTCATATTA	1133
	TAATATGAATTTCTCTCTTCAACTAAACAATGTACATGAACATA CCTTTCCAATTTTTCA**G**ATAGATTGTCAGCAGAATCAACAGAA GGGATTTGGTATATGTCTGACAATTCCAGGCGCT	1134
	CAATCTAT**C**TGAAAAAT	1135
	ATTTTTCA**G**ATAGATTG	1136
Congenital absence of vas deferens Glu56Lys GAA-AAA	AGGACAACTAAAATATTTGCACATGCAACTTATTGGTCCCACT TTTTATTCTTTTGCAGA**G**AATGGGATAGAGAGCTGGCTTCAAA GAAAAATCCTAAACTCATTAATGCCCTTCGGCGAT	1137
	ATCGCCGAAGGGCATTAATGAGTTTAGGATTTTTCTTTGAAGC CAGCTCTCTATCCCATT**C**TCTGCAAAAGAATAAAAAGTGGGA CCAATAAGTTGCATGTGCAAATATTTTAGTTGTCCT	1138
	TTTGCAGA**G**AATGGGAT	1139
	ATCCCATT**C**TCTGCAAA	1140

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Trp57Gly TGG to GGG	AGGACAACTAAAATATTTGCACATGCAACTTATTGGTCCCACT TTTTATTCTTTTGCAGA**G**AATGGGATAGAGAGCTGGCTTCAAA GAAAAATCCTAAACTCATTAATGCCCTTCGGCGAT	1141
	ATCGCCGAAGGGCATTAATGAGTTTAGGATTTTTCTTTGAAGC CAGCTCTCTATCCCATT**C**TCTGCAAAAGAATAAAAAGTGGGA CCAATAAGTTGCATGTGCAAATATTTTAGTTGTCCT	1142
	TTTGCAGA**G**AATGGGAT	1143
	ATCCCATT**C**TCTGCAAA	1144
Cystic fibrosis Trp57Term TGG to TGA	AACTAAAATATTTGCACATGCAACTTATTGGTCCCACTTTTTAT TCTTTTGCAGAGAATG**G**GATAGAGAGCTGGCTTCAAAGAAAA ATCCTAAACTCATTAATGCCCTTCGGCGATGTTTT	1145
	AAAACATCGCCGAAGGGCATTAATGAGTTTAGGATTTTTCTTT GAAGCCAGCTCTCTATC**C**CATTCTCTGCAAAAGAATAAAAAGT GGGACCAATAAGTTGCATGTGCAAATATTTTAGTT	1146
	AGAGAATG**G**GATAGAGA	1147
	TCTCTATC**C**CATTCTCT	1148
Congenital absence of vas deferens Asp58Asn GAT to AAT	ACTAAAATATTTGCACATGCAACTTATTGGTCCCACTTTTTATT CTTTTGCAGAGAATGG**G**ATAGAGAGCTGGCTTCAAAGAAAAA TCCTAAACTCATTAATGCCCTTCGGCGATGTTTTT	1149
	AAAAACATCGCCGAAGGGCATTAATGAGTTTAGGATTTTTCTT TGAAGCCAGCTCTCTAT**C**CCATTCTCTGCAAAAGAATAAAAAG TGGGACCAATAAGTTGCATGTGCAAATATTTTAGT	1150
	GAGAATGG**G**ATAGAGAG	1151
	CTCTCTAT**C**CCATTCTC	1152
Cystic fibrosis Glu60Term GAG to TAG	ATATTTGCACATGCAACTTATTGGTCCCACTTTTTATTCTTTTG CAGAGAATGGGATAGA**G**AGCTGGCTTCAAAGAAAAATCCTAA ACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGA	1153
	TCCAGAAAAAACATCGCCGAAGGGCATTAATGAGTTTAGGAT TTTTCTTTGAAGCCAGCT**C**TCTATCCCATTCTCTGCAAAAGAA TAAAAAGTGGGACCAATAAGTTGCATGTGCAAATAT	1154
	GGGATAGA**G**AGCTGGCT	1155
	AGCCAGCT**C**TCTATCCC	1156

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Pro67Leu CCT to CTT	GGTCCCACTTTTTATTCTTTTGCAGAGAATGGGATAGAGAGC TGGCTTCAAAGAAAAATC**C**TAAACTCATTAATGCCCTTCGGC GATGTTTTTTCTGGAGATTTATGTTCTATGGAATCTT	1157
	AAGATTCCATAGAACATAAATCTCCAGAAAAAACATCGCCGAA GGGCATTAATGAGTTTA**G**GATTTTTCTTTGAAGCCAGCTCTCT ATCCCATTCTCTGCAAAAGAATAAAAAGTGGGACC	1158
	GAAAAATC**C**TAAACTCA	1159
	TGAGTTTA**G**GATTTTTC	1160
Cystic fibrosis Arg74Trp CGG to TGG	TGCAGAGAATGGGATAGAGAGCTGGCTTCAAAGAAAAATCCT AAACTCATTAATGCCCTT**C**GGCGATGTTTTTTCTGGAGATTTA TGTTCTATGGAATCTTTTTATATTTAGGGGTAAGGA	1161
	TCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAAATCT CCAGAAAAAACATCGCC**G**AAGGGCATTAATGAGTTTAGGATT TTTCTTTGAAGCCAGCTCTCTATCCCATTCTCTGCA	1162
	ATGCCCTT**C**GGCGATGT	1163
	ACATCGCC**G**AAGGGCAT	1164
Congenital absence of vas deferens ARG75GLN CGA to CAA	GAGAATGGGATAGAGAGCTGGCTTCAAAGAAAAATCCTAAAC TCATTAATGCCCTTCGGC**G**ATGTTTTTTCTGGAGATTTATGTT CTATGGAATCTTTTTATATTTAGGGGTAAGGATCTC	1165
	GAGATCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAA ATCTCCAGAAAAAACAT**C**GCCGAAGGGCATTAATGAGTTTAG GATTTTTCTTTGAAGCCAGCTCTCTATCCCATTCTC	1166
	CCTTCGGC**G**ATGTTTTT	1167
	AAAAACAT**C**GCCGAAGG	1168
Cystic fibrosis Arg75Leu CGA to CTA	GAGAATGGGATAGAGAGCTGGCTTCAAAGAAAAATCCTAAAC TCATTAATGCCCTTCGGC**G**ATGTTTTTTCTGGAGATTTATGTT CTATGGAATCTTTTTATATTTAGGGGTAAGGATCTC	1169
	GAGATCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAA ATCTCCAGAAAAAACAT**C**GCCGAAGGGCATTAATGAGTTTAG GATTTTTCTTTGAAGCCAGCTCTCTATCCCATTCTC	1170
	CCTTCGGC**G**ATGTTTTT	1171
	AAAAACAT**C**GCCGAAGG	1172
Cystic fibrosis Arg75Term CGA to TGA	AGAGAATGGGATAGAGAGCTGGCTTCAAAGAAAAATCCTAAA CTCATTAATGCCCTTCGG**C**GATGTTTTTTCTGGAGATTTATGT TCTATGGAATCTTTTTATATTTAGGGGTAAGGATCT	1173

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGATCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAAA TCTCCAGAAAAAACATCGCCGAAGGGCATTAATGAGTTTAGG ATTTTTCTTTGAAGCCAGCTCTCTATCCCATTCTCT	1174
	CCCTTCGGCGATGTTTT	1175
	AAAACATCGCCGAAGGG	1176
Cystic fibrosis Gly85Glu GGA to GAA	AAAATCCTAAACTCATTAATGCCCTTCGGCGATGTTTTTTCTG GAGATTTATGTTCTATGGAATCTTTTTATATTTAGGGGTAAGG ATCTCATTTGTACATTCATTATGTATCACATAACT	1177
	AGTTATGTGATACATAATGAATGTACAAATGAGATCCTTACCC CTAAATATAAAAAGATTCCATAGAACATAAATCTCCAGAAAAA ACATCGCCGAAGGGCATTAATGAGTTTAGGATTTT	1178
	GTTCTATGGAATCTTTT	1179
	AAAAGATTCCATAGAAC	1180
Cystic fibrosis Gly85Val GGA to GTA	AAAATCCTAAACTCATTAATGCCCTTCGGCGATGTTTTTTCTG GAGATTTATGTTCTATGGAATCTTTTTATATTTAGGGGTAAGG ATCTCATTTGTACATTCATTATGTATCACATAACT	1181
	AGTTATGTGATACATAATGAATGTACAAATGAGATCCTTACCC CTAAATATAAAAAGATTCCATAGAACATAAATCTCCAGAAAAA ACATCGCCGAAGGGCATTAATGAGTTTAGGATTTT	1182
	GTTCTATGGAATCTTTT	1183
	AAAAGATTCCATAGAAC	1184
Cystic fibrosis Leu88Ser TTA to TCA	AACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGATTTAT GTTCTATGGAATCTTTTTATATTTAGGGGTAAGGATCTCATTT GTACATTCATTATGTATCACATAACTATATGCATT	1185
	AATGCATATAGTTATGTGATACATAATGAATGTACAAATGAGA TCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAAATCT CCAGAAAAAAACATCGCCGAAGGGCATTAATGAGTT	1186
	AATCTTTTTATATTTAG	1187
	CTAAATATAAAAAGATT	1188
Cystic fibrosis Phe87Leu TTT to CTT	CCTAAACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGAT TTATGTTCTATGGAATCTTTTTATATTTAGGGGTAAGGATCTC ATTTGTACATTCATTATGTATCACATAACTATATG	1189
	CATATAGTTATGTGATACATAATGAATGTACAAATGAGATCCT TACCCCTAAATATAAAAAGATTCCATAGAACATAAATCTCCAG AAAAAACATCGCCGAAGGGCATTAATGAGTTTAGG	1190

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATGGAATCTTTTTATAT	1191
	ATATAAAAAGATTCCAT	1192
Cystic fibrosis Leu88Term TTA to TGA	AACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGATTTAT GTTCTATGGAATCTTTTTATATTTAGGGGTAAGGATCTCATTT GTACATTCATTATGTATCACATAACTATATGCATT	1193
	AATGCATATAGTTATGTGATACATAATGAATGTACAAATGAGA TCCTTACCCCTAAATATAAAAAAGATTCCATAGAACATAAATCT CCAGAAAAAACATCGCCGAAGGGCATTAATGAGTT	1194
	AATCTTTTTATATTTAG	1195
	CTAAATATAAAAAAGATT	1196
Cystic fibrosis Leu88Term TTA to TAA	AACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGATTTAT GTTCTATGGAATCTTTTTATATTTAGGGGTAAGGATCTCATTT GTACATTCATTATGTATCACATAACTATATGCATT	1197
	AATGCATATAGTTATGTGATACATAATGAATGTACAAATGAGA TCCTTACCCCTAAATATAAAAAAGATTCCATAGAACATAAATCT CCAGAAAAAACATCGCCGAAGGGCATTAATGAGTT	1198
	AATCTTTTTATATTTAG	1199
	CTAAATATAAAAAAGATT	1200
Cystic fibrosis Gly91Arg GGG to AGG	AATGCCCTTCGGCGATGTTTTTTCTGGAGATTTATGTTCTATG GAATCTTTTTATATTTAGGGGTAAGGATCTCATTTGTACATTC ATTATGTATCACATAACTATATGCATTTTTGTGAT	1201
	ATCACAAAAATGCATATAGTTATGTGATACATAATGAATGTAC AAATGAGATCCTTACCCCTAAATATAAAAAAGATTCCATAGAAC ATAAATCTCCAGAAAAAACATCGCCGAAGGGCATT	1202
	TATATTTAGGGGTAAGG	1203
	CCTTACCCCTAAATATA	1204
Cystic fibrosis Gln98Arg CAG to CGG	AATAAATGAAATTTAATTTCTCTGTTTTTCCCCTTTTGTAGGAA GTCACCAAAGCAGTACAGCCTCTCTTACTGGGAAGAATCATA GCTTCCTATGACCCGGATAACAAGGAGGAACGCTC	1205
	GAGCGTTCCTCCTTGTTATCCGGGTCATAGGAAGCTATGATT CTTCCCAGTAAGAGAGGCTGTACTGCTTTGGTGACTTCCTAC AAAAGGGGAAAAACAGAGAAATTAAATTTCATTTATT	1206
	AGCAGTACAGCCTCTCT	1207
	AGAGAGGCTGTACTGCT	1208

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Gln98Term CAG-TAG	AAATAAATGAAATTTAATTTCTCTGTTTTTCCCCTTTTGTAGGA AGTCACCAAAGCAGTA**C**AGCCTCTCTTACTGGGAAGAATCAT AGCTTCCTATGACCCGGATAACAAGGAGGAACGCT	1209
	AGCGTTCCTCCTTGTTATCCGGGTCATAGGAAGCTATGATTC TTCCCAGTAAGAGAGGCT**G**TACTGCTTTGGTGACTTCCTACA AAAGGGGAAAAACAGAGAAATTAAATTTCATTTATTT	1210
	AAGCAGTA**C**AGCCTCTC	1211
	GAGAGGCT**G**TACTGCTT	1212
Cystic fibrosis Ser108Phe TCC to TTC	CCCTTTTGTAGGAAGTCACCAAAGCAGTACAGCCTCTCTTAC TGGGAAGAATCATAGCTT**C**CTATGACCCGGATAACAAGGAGG AACGCTCTATCGCGATTTATCTAGGCATAGGCTTATG	1213
	CATAAGCCTATGCCTAGATAAATCGCGATAGAGCGTTCCTCC TTGTTATCCGGGTCATAG**G**AAGCTATGATTCTTCCCAGTAAG AGAGGCTGTACTGCTTTGGTGACTTCCTACAAAAGGG	1214
	CATAGCTT**C**CTATGACC	1215
	GGTCATAG**G**AAGCTATG	1216
Cystic fibrosis Tyr109Cys TAT to TGT	TTTTGTAGGAAGTCACCAAAGCAGTACAGCCTCTCTTACTGG GAAGAATCATAGCTTCCT**A**TGACCCGGATAACAAGGAGGAAC GCTCTATCGCGATTTATCTAGGCATAGGCTTATGCCT	1217
	AGGCATAAGCCTATGCCTAGATAAATCGCGATAGAGCGTTCC TCCTTGTTATCCGGGTCA**T**AGGAAGCTATGATTCTTCCCAGT AAGAGAGGCTGTACTGCTTTGGTGACTTCCTACAAAA	1218
	AGCTTCCT**A**TGACCCGG	1219
	CCGGGTCA**T**AGGAAGCT	1220
Cystic fibrosis Asp110His GAC to CAC	TTGTAGGAAGTCACCAAAGCAGTACAGCCTCTCTTACTGGGA AGAATCATAGCTTCCTAT**G**ACCCGGATAACAAGGAGGAACGC TCTATCGCGATTTATCTAGGCATAGGCTTATGCCTTC	1221
	GAAGGCATAAGCCTATGCCTAGATAAATCGCGATAGAGCGTT CCTCCTTGTTATCCGGGT**C**ATAGGAAGCTATGATTCTTCCCA GTAAGAGAGGCTGTACTGCTTTGGTGACTTCCTACAA	1222
	CTTCCTAT**G**ACCCGGAT	1223
	ATCCGGGT**C**ATAGGAAG	1224

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Congenital absence of vas deferens Pro111Leu CCG to CTG	AGGAAGTCACCAAAGCAGTACAGCCTCTCTTACTGGGAAGAA TCATAGCTTCCTATGACC**C**GGATAACAAGGAGGAACGCTCTA TCGCGATTTATCTAGGCATAGGCTTATGCCTTCTCTT	1225
	AAGAGAAGGCATAAGCCTATGCCTAGATAAATCGCGATAGAG CGTTCCTCCTTGTTATCC**G**GGTCATAGGAAGCTATGATTCTT CCCAGTAAGAGAGGCTGTACTGCTTTGGTGACTTCCT	1226
	CTATGACC**C**GGATAACA	1227
	TGTTATCC**G**GGTCATAG	1228
Cystic fibrosis Arg117Cys CGC to TGC	GTACAGCCTCTCTTACTGGGAAGAATCATAGCTTCCTATGAC CCGGATAACAAGGAGGAA**C**GCTCTATCGCGATTTATCTAGGC ATAGGCTTATGCCTTCTCTTTATTGTGAGGACACTGC	1229
	GCAGTGTCCTCACAATAAAGAGAAGGCATAAGCCTATGCCTA GATAAATCGCGATAGAGC**G**TTCCTCCTTGTTATCCGGGTCAT AGGAAGCTATGATTCTTCCCAGTAAGAGAGGCTGTAC	1230
	AGGAGGAA**C**GCTCTATC	1231
	GATAGAGC**G**TTCCTCCT	1232
Cystic fibrosis Arg117His CGC to CAC	TACAGCCTCTCTTACTGGGAAGAATCATAGCTTCCTATGACC CGGATAACAAGGAGGAAC**G**CTCTATCGCGATTTATCTAGGCA TAGGCTTATGCCTTCTCTTTATTGTGAGGACACTGCT	1233
	AGCAGTGTCCTCACAATAAAGAGAAGGCATAAGCCTATGCCT AGATAAATCGCGATAGAG**C**GTTCCTCCTTGTTATCCGGGTCA TAGGAAGCTATGATTCTTCCCAGTAAGAGAGGCTGTA	1234
	GGAGGAAC**G**CTCTATCG	1235
	CGATAGAG**C**GTTCCTCC	1236
Cystic fibrosis Arg117Leu CGC to CTC	TACAGCCTCTCTTACTGGGAAGAATCATAGCTTCCTATGACC CGGATAACAAGGAGGAAC**G**CTCTATCGCGATTTATCTAGGCA TAGGCTTATGCCTTCTCTTTATTGTGAGGACACTGCT	1237
	AGCAGTGTCCTCACAATAAAGAGAAGGCATAAGCCTATGCCT AGATAAATCGCGATAGAG**C**GTTCCTCCTTGTTATCCGGGTCA TAGGAAGCTATGATTCTTCCCAGTAAGAGAGGCTGTA	1238
	GGAGGAAC**G**CTCTATCG	1239
	CGATAGAG**C**GTTCCTCC	1240

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Arg117Pro CGC to CCC	TACAGCCTCTCTTACTGGGAAGAATCATAGCTTCCTATGACC CGGATAACAAGGAGGAAC**G**CTCTATCGCGATTTATCTAGGCA TAGGCTTATGCCTTCTCTTTATTGTGAGGACACTGCT	1241
	AGCAGTGTCCTCACAATAAAGAGAAGGCATAAGCCTATGCCT AGATAAATCGCGATAGAG**C**GTTCCTCCTTGTTATCCGGGTCA TAGGAAGCTATGATTCTTCCCAGTAAGAGAGGCTGTA	1242
	GGAGGAAC**G**CTCTATCG	1243
	CGATAGAG**C**GTTCCTCC	1244
Cystic fibrosis Ala120Thr GCG-ACG	CTCTTACTGGGAAGAATCATAGCTTCCTATGACCCGGATAAC AAGGAGGAACGCTCTATC**G**CGATTTATCTAGGCATAGGCTTA TGCCTTCTCTTTATTGTGAGGACACTGCTCCTACACC	1245
	GGTGTAGGAGCAGTGTCCTCACAATAAAGAGAAGGCATAAG CCTATGCCTAGATAAATCG**C**GATAGAGCGTTCCTCCTTGTTA TCCGGGTCATAGGAAGCTATGATTCTTCCCAGTAAGAG	1246
	GCTCTATC**G**CGATTTAT	1247
	ATAAATCG**C**GATAGAGC	1248
Cystic fibrosis Tyr122Term TAT to TAA	GGGAAGAATCATAGCTTCCTATGACCCGGATAACAAGGAGGA ACGCTCTATCGCGATTTA**T**CTAGGCATAGGCTTATGCCTTCT CTTTATTGTGAGGACACTGCTCCTACACCCAGCCATT	1249
	AATGGCTGGGTGTAGGAGCAGTGTCCTCACAATAAAGAGAA GGCATAAGCCTATGCCTAG**A**TAAATCGCGATAGAGCGTTCCT CCTTGTTATCCGGGTCATAGGAAGCTATGATTCTTCCC	1250
	GCGATTTA**T**CTAGGCAT	1251
	ATGCCTAG**A**TAAATCGC	1252
Cystic fibrosis Gly126Asp GGC-GAC	TAGCTTCCTATGACCCGGATAACAAGGAGGAACGCTCTATCG CGATTTATCTAGGCATAG**G**CTTATGCCTTCTCTTTATTGTGAG GACACTGCTCCTACACCCAGCCATTTTTGGCCTTCA	1253
	TGAAGGCCAAAAATGGCTGGGTGTAGGAGCAGTGTCCTCAC AATAAAGAGAAGGCATAAG**C**CTATGCCTAGATAAATCGCGAT AGAGCGTTCCTCCTTGTTATCCGGGTCATAGGAAGCTA	1254
	AGGCATAG**G**CTTATGCC	1255
	GGCATAAG**C**CTATGCCT	1256

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis His139Arg CAC to CGC	TCGCGATTTATCTAGGCATAGGCTTATGCCTTCTCTTTATTGT GAGGACACTGCTCCTACACCCAGCCATTTTTGGCCTTCATCA CATTGGAATGCAGATGAGAATAGCTATGTTTAGTTT	1257
	AAACTAAACATAGCTATTCTCATCTGCATTCCAATGTGATGAA GGCCAAAAATGGCTGGGTGTAGGAGCAGTGTCCTCACAATA AAGAGAAGGCATAAGCCTATGCCTAGATAAATCGCGA	1258
	GCTCCTACACCCAGCCA	1259
	TGGCTGGGTGTAGGAGC	1260
Cystic fibrosis Ala141Asp GCC to GAC	TTTATCTAGGCATAGGCTTATGCCTTCTCTTTATTGTGAGGAC ACTGCTCCTACACCCAGCCATTTTTGGCCTTCATCACATTGG AATGCAGATGAGAATAGCTATGTTTAGTTTGATTTA	1261
	TAAATCAAACTAAACATAGCTATTCTCATCTGCATTCCAATGT GATGAAGGCCAAAAATGGCTGGGTGTAGGAGCAGTGTCCTC ACAATAAAGAGAAGGCATAAGCCTATGCCTAGATAAA	1262
	ACACCCAGCCATTTTTG	1263
	CAAAAATGGCTGGGTGT	1264
Cystic fibrosis Ile148Thr ATT to ACT	GCCTTCTCTTTATTGTGAGGACACTGCTCCTACACCCAGCCA TTTTTGGCCTTCATCACATTGGAATGCAGATGAGAATAGCTAT GTTTAGTTTGATTTATAAGAAGGTAATACTTCCTTG	1265
	CAAGGAAGTATTACCTTCTTATAAATCAAACTAAACATAGCTA TTCTCATCTGCATTCCAATGTGATGAAGGCCAAAAATGGCTG GGTGTAGGAGCAGTGTCCTCACAATAAAGAGAAGGC	1266
	TCATCACATTGGAATGC	1267
	GCATTCCAATGTGATGA	1268
Cystic fibrosis Gly149Arg GGA to AGA	CTTCTCTTTATTGTGAGGACACTGCTCCTACACCCAGCCATTT TTGGCCTTCATCACATTGGAATGCAGATGAGAATAGCTATGTT TAGTTTGATTTATAAGAAGGTAATACTTCCTTGCA	1269
	TGCAAGGAAGTATTACCTTCTTATAAATCAAACTAAACATAGC TATTCTCATCTGCATTCCAATGTGATGAAGGCCAAAAATGGCT GGGTGTAGGAGCAGTGTCCTCACAATAAAGAGAAG	1270
	ATCACATTGGAATGCAG	1271
	CTGCATTCCAATGTGAT	1272

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Gln151Term CAG to TAG	TTTATTGTGAGGACACTGCTCCTACACCCAGCCATTTTTGGC CTTCATCACATTGGAATG**C**AGATGAGAATAGCTATGTTTAGTT TGATTTATAAGAAGGTAATACTTCCTTGCACAGGCC	1273
	GGCCTGTGCAAGGAAGTATTACCTTCTTATAAATCAAACTAAA CATAGCTATTCTCATCT**G**CATTCCAATGTGATGAAGGCCAAAA ATGGCTGGGTGTAGGAGCAGTGTCCTCACAATAAA	1274
	TTGGAATG**C**AGATGAGA	1275
	TCTCATCT**G**CATTCCAA	1276
Cystic fibrosis Lys166Glu AAG-GAG	AATATATTTGTATTTTGTTTGTTGAAATTATCTAACTTTCCATTT TTCTTTTAGACTTTA**A**AGCTGTCAAGCCGTGTTCTAGATAAAA TAAGTATTGGACAACTTGTTAGTCTCCTTTCCA	1277
	TGGAAAGGAGACTAACAAGTTGTCCAATACTTATTTTATCTAG AACACGGCTTGACAGCT**T**TAAAGTCTAAAAGAAAAATGGAAA GTTAGATAATTTCAACAAACAAAATACAAATATATT	1278
	AGACTTTA**A**AGCTGTCA	1279
	TGACAGCT**T**TAAAGTCT	1280
Cystic fibrosis Ile175Val ATA-GTA	TTATCTAACTTTCCATTTTTCTTTTAGACTTTAAAGCTGTCAAG CCGTGTTCTAGATAAA**A**TAAGTATTGGACAACTTGTTAGTCTC CTTTCCAACAACCTGAACAAATTTGATGAAGTAT	1281
	ATACTTCATCAAATTTGTTCAGGTTGTTGGAAAGGAGACTAAC AAGTTGTCCAATACTTA**T**TTTATCTAGAACACGGCTTGACAGC TTTAAAGTCTAAAAGAAAAATGGAAAGTTAGATAA	1282
	TAGATAAA**A**TAAGTATT	1283
	AATACTTA**T**TTTATCTA	1284
Cystic fibrosis Gly178Arg GGA to AGA	TTTCCATTTTTCTTTTAGACTTTAAAGCTGTCAAGCCGTGTTCT AGATAAAATAAGTATT**G**GACAACTTGTTAGTCTCCTTTCCAAC AACCTGAACAAATTTGATGAAGTATGTACCTATT	1285
	AATAGGTACATACTTCATCAAATTTGTTCAGGTTGTTGGAAAG GAGACTAACAAGTTGTC**C**AATACTTATTTTATCTAGAACACGG CTTGACAGCTTTAAAGTCTAAAAGAAAAATGGAAA	1286
	TAAGTATT**G**GACAACTT	1287
	AAGTTGTC**C**AATACTTA	1288
Cystic fibrosis His199Gln CAT to CAG	AAGATACAATGACACCTGTTTTTGCTGTGCTTTTATTTTCCAG GGACTTGCATTGGCACA**T**TTCGTGTGGATCGCTCCTTTGCAA GTGGCACTCCTCATGGGGCTAATCTGGGAGTTGTTA	1289

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAACAACTCCCAGATTAGCCCCATGAGGAGTGCCACTTGCAA AGGAGCGATCCACACGAAATGTGCCAATGCAAGTCCCTGGA AAATAAAAGCACAGCAAAAACAGGTGTCATTGTATCTT	1290
	TTGGCACATTTCGTGTG	1291
	CACACGAAATGTGCCAA	1292
Cystic fibrosis His199Tyr CAT to TAT	GGAAGATACAATGACACCTGTTTTTGCTGTGCTTTTATTTTCC AGGGACTTGCATTGGCACATTTCGTGTGGATCGCTCCTTTGC AAGTGGCACTCCTCATGGGGCTAATCTGGGAGTTGT	1293
	ACAACTCCCAGATTAGCCCCATGAGGAGTGCCACTTGCAAAG GAGCGATCCACACGAAATGTGCCAATGCAAGTCCCTGGAAA ATAAAAGCACAGCAAAAACAGGTGTCATTGTATCTTCC	1294
	CATTGGCACATTTCGTG	1295
	CACGAAATGTGCCAATG	1296
Cystic fibrosis Pro205Ser CCT to TCT	TGTTTTTGCTGTGCTTTTATTTTCCAGGGACTTGCATTGGCAC ATTTCGTGTGGATCGCTCCTTTGCAAGTGGCACTCCTCATGG GGCTAATCTGGGAGTTGTTACAGGCGTCTGCCTTCT	1297
	AGAAGGCAGACGCCTGTAACAACTCCCAGATTAGCCCCATG AGGAGTGCCACTTGCAAAGGAGCGATCCACACGAAATGTGC CAATGCAAGTCCCTGGAAAATAAAAGCACAGCAAAAACA	1298
	GGATCGCTCCTTTGCAA	1299
	TTGCAAAGGAGCGATCC	1300
Cystic fibrosis Leu206Trp TTG to TGG	TTTGCTGTGCTTTTATTTTCCAGGGACTTGCATTGGCACATTT CGTGTGGATCGCTCCTTTGCAAGTGGCACTCCTCATGGGGC TAATCTGGGAGTTGTTACAGGCGTCTGCCTTCTGTGG	1301
	CCACAGAAGGCAGACGCCTGTAACAACTCCCAGATTAGCCC CATGAGGAGTGCCACTTGCAAAGGAGCGATCCACACGAAAT GTGCCAATGCAAGTCCCTGGAAAATAAAAGCACAGCAAA	1302
	CGCTCCTTTGCAAGTGG	1303
	CCACTTGCAAAGGAGCG	1304
Cystic fibrosis Gln220Term CAG to TAG	TTCGTGTGGATCGCTCCTTTGCAAGTGGCACTCCTCATGGG GCTAATCTGGGAGTTGTTACAGGCGTCTGCCTTCTGTGGACT TGGTTTCCTGATAGTCCTTGCCCTTTTTCAGGCTGGGC	1305
	GCCCAGCCTGAAAAAGGGCAAGGACTATCAGGAAACCAAGT CCACAGAAGGCAGACGCCTGTAACAACTCCCAGATTAGCCC CATGAGGAGTGCCACTTGCAAAGGAGCGATCCACACGAA	1306

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGTTGTTACAGGCGTCT	1307
	AGACGCCTGTAACAACT	1308
Cystic fibrosis Cys225Arg TGT-CGT	CCTTTGCAAGTGGCACTCCTCATGGGGCTAATCTGGGAGTT GTTACAGGCGTCTGCCTTCTGTGGACTTGGTTTCCTGATAGT CCTTGCCCTTTTTCAGGCTGGGCTAGGGAGAATGATGA	1309
	TCATCATTCTCCCTAGCCCAGCCTGAAAAAGGGCAAGGACTA TCAGGAAACCAAGTCCACAGAAGGCAGACGCCTGTAACAAC TCCCAGATTAGCCCCATGAGGAGTGCCACTTGCAAAGG	1310
	CTGCCTTCTGTGGACTT	1311
	AAGTCCACAGAAGGCAG	1312
Cystic fibrosis Val232Asp GTC to GAC	TGGGGCTAATCTGGGAGTTGTTACAGGCGTCTGCCTTCTGT GGACTTGGTTTCCTGATAGTCCTTGCCCTTTTTCAGGCTGGG CTAGGGAGAATGATGATGAAGTACAGGTAGCAACCTAT	1313
	ATAGGTTGCTACCTGTACTTCATCATCATTCTCCCTAGCCCA GCCTGAAAAAGGGCAAGGACTATCAGGAAACCAAGTCCACA GAAGGCAGACGCCTGTAACAACTCCCAGATTAGCCCCA	1314
	CCTGATAGTCCTTGCCC	1315
	GGGCAAGGACTATCAGG	1316
Cystic fibrosis Gly239Arg GGG to AGG	GTTACAGGCGTCTGCCTTCTGTGGACTTGGTTTCCTGATAGT CCTTGCCCTTTTTCAGGCTGGGCTAGGGAGAATGATGATGAA GTACAGGTAGCAACCTATTTTCATAACTTGAAAGTTT	1317
	AAACTTTCAAGTTATGAAAATAGGTTGCTACCTGTACTTCATC ATCATTCTCCCTAGCCCAGCCTGAAAAAGGGCAAGGACTATC AGGAAACCAAGTCCACAGAAGGCAGACGCCTGTAAC	1318
	TTTCAGGCTGGGCTAGG	1319
	CCTAGCCCAGCCTGAAA	1320

EXAMPLE 10
Cyclin-dependent kinase inhibitor 2A - CDKN2A

The human CDKN2A gene was also designated MTS-1 for multiple tumor suppressor-1 and has been implicated in multiple cancers including, for example, malignant melanoma. Malignant melanoma is a cutaneous neoplasm of melanocytes. Melanomas generally have features of asymmetry, irregular border, variegated color, and diameter greater than 6 mm. The precise cause of melanoma is

unknown, but sunlight and heredity are risk factors. Melanoma has been increasing during the past few decades.

The CDKN2A gene has been found to be homozygously deleted at high frequency in cell lines derived from tumors of lung, breast, brain, bone, skin, bladder, kidney, ovary, and lymphocyte. Melanoma cell lines carried at least one copy of CDKN2A in combination with a deleted allele. Melanoma cell lines that carried at least 1 copy of CDKN2A frequently showed nonsense, missense, or frameshift mutations in the gene. Thus, CDKN2A may rival p53 (see Example 5) in the universality of its involvement in tumorigenesis. The attached table discloses the correcting oligonucleotide base sequences for the CDKN2A oligonucleotides of the invention.

Table 17
CDKN2A Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Melanoma Trp15Term TGG-TAG	GGGCGGCGGGGAGCAGCATGGAGCCGGCGGCGGGGAGCAG CATGGAGCCTTCGGCTGACT**G**GCTGGCCACGGCCGCGGCCC GGGGTCGGGTAGAGGAGGTGCGGGCGCTGCTGGAGGCGGG	1321
	CCCGCCTCCAGCAGCGCCCGCACCTCCTCTACCCGACCCCG GGCCGCGGCCGTGGCCAGC**C**AGTCAGCCGAAGGCTCCATGC TGCTCCCCGCCGCCGGCTCCATGCTGCTCCCCGCCGCCC	1322
	GGCTGACT**G**GCTGGCCA	1323
	TGGCCAGC**C**AGTCAGCC	1324
Melanoma Leu16Pro CTG-CCG	CGGCGGGGAGCAGCATGGAGCCGGCGGCGGGGAGCAGCAT GGAGCCTTCGGCTGACTGGC**T**GGCCACGGCCGCGGCCCGG GGTCGGGTAGAGGAGGTGCGGGCGCTGCTGGAGGCGGGGG C	1325
	GCCCCCGCCTCCAGCAGCGCCCGCACCTCCTCTACCCGACC CCGGGCCGCGGCCGTGGCC**A**GCCAGTCAGCCGAAGGCTCC ATGCTGCTCCCCGCCGCCGGCTCCATGCTGCTCCCCGCCG	1326
	TGACTGGC**T**GGCCACGG	1327
	CCGTGGCC**A**GCCAGTCA	1328
Melanoma Gly23Asp GGT-GAT	CGGCGGCGGGGAGCAGCATGGAGCCTTCGGCTGACTGGCTG GCCACGGCCGCGGCCCGGG**G**TCGGGTAGAGGAGGTGCGGG CGCTGCTGGAGGCGGGGGCGCTGCCCAACGCACCGAATAG	1329
	CTATTCGGTGCGTTGGGCAGCGCCCCCGCCTCCAGCAGCGC CCGCACCTCCTCTACCCGA**C**CCCGGGCCGCGGCC**G**TGGCCA GCCAGTCAGCCGAAGGCTCCATGCTGCTCCCCGCCGCCG	1330
	GGCCCGGG**G**TCGGGTAG	1331

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTACCCGACCCCGGGCC	1332
Melanoma Arg24Pro CGG-CCG	CGGCGGGGAGCAGCATGGAGCCTTCGGCTGACTGGCTGGCC ACGGCCGCGGCCCGGGGTCGGGTAGAGGAGGTGCGGGCGC TGCTGGAGGCGGGGGCGCTGCCCAACGCACCGAATAGTTA	1333
	TAACTATTCGGTGCGTTGGGCAGCGCCCCCGCCTCCAGCAGC GCCCGCACCTCCTCTACCCGACCCCGGGCCGCGGCCGTGGC CAGCCAGTCAGCCGAAGGCTCCATGCTGCTCCCCGCCG	1334
	CCGGGGTCGGGTAGAGG	1335
	CCTCTACCCGACCCCGG	1336
Melanoma Leu32Pro CTG-CCG	CGGCTGACTGGCTGGCCACGGCCGCGGCCCGGGGTCGGGT AGAGGAGGTGCGGGCGCTGCTGGAGGCGGGGGCGCTGCCC AACGCACCGAATAGTTACGGTCGGAGGCCGATCCAGGTGGG	1337
	CCCACCTGGATCGGCCTCCGACCGTAACTATTCGGTGCGTTG GGCAGCGCCCCCGCCTCCAGCAGCGCCCGCACCTCCTCTAC CCGACCCCGGGCCGCGGCCGTGGCCAGCCAGTCAGCCG	1338
	GGCGCTGCTGGAGGCGG	1339
	CCGCCTCCAGCAGCGCC	1340
Melanoma Gly35Ala GGG-GCG	GGCTGGCCACGGCCGCGGCCCGGGGTCGGGTAGAGGAGGT GCGGGCGCTGCTGGAGGCGGGGGCGCTGCCCAACGCACCG AATAGTTACGGTCGGAGGCCGATCCAGGTGGGTAGAGGGTC	1341
	GACCCTCTACCCACCTGGATCGGCCTCCGACCGTAACTATTC GGTGCGTTGGGCAGCGCCCCCGCCTCCAGCAGCGCCCGCAC CTCCTCTACCCGACCCCGGGCCGCGGCCGTGGCCAGCC	1342
	GGAGGCGGGGGCGCTGC	1343
	GCAGCGCCCCCGCCTCC	1344
Melanoma Tyr44Term TACg-TAA	GGTAGAGGAGGTGCGGGCGCTGCTGGAGGCGGGGGCGCTG CCCAACGCACCGAATAGTTACGGTCGGAGGCCGATCCAGGTG GGTAGAGGGTCTGCAGCGGGAGCAGGGGATGGCGGGCGA	1345
	TCGCCCGCCATCCCCTGCTCCCGCTGCAGACCCTCTACCCAC CTGGATCGGCCTCCGACCGTAACTATTCGGTGCGTTGGGCAG CGCCCCCGCCTCCAGCAGCGCCCGCACCTCCTCTACC	1346
	AATAGTTACGGTCGGAG	1347
	CTCCGACCGTAACTATT	1348
Melanoma Met53Ile ATGa-ATC	TCTCCCATACCTGCCCCCACCCTGGCTCTGACCACTCTGCTC TCTCTGGCAGGTCATGATGATGGGCAGCGCCCGCGTGGCGG AGCTGCTGCTGCTCCACGGCGCGGAGCCCAACTGCGCA	1349
	TGCGCAGTTGGGCTCCGCGCCGTGGAGCAGCAGCAGCTCCG CCACGCGGGCGCTGCCCATCATCATGACCTGCCAGAGAGAG CAGAGTGGTCAGAGCCAGGGTGGGGGCAGGTATGGGAGA	1350
	GTCATGATGATGGGCAG	1351

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTGCCCATCATCATGAC	1352
Melanoma Met54Ile ATGg-ATT	CCCATACCTGCCCCCACCCTGGCTCTGACCACTCTGCTCTCT CTGGCAGGTCATGATGATGGGCAGCGCCCGCGTGGCGGAGC TGCTGCTGCTCCACGGCGCGGAGCCCAACTGCGCAGAC	1353
	GTCTGCGCAGTTGGGCTCCGCGCCGTGGAGCAGCAGCAGCT CCGCCACGCGGGCGCTGCCCATCATCATGACCTGCCAGAGA GAGCAGAGTGGTCAGAGCCAGGGTGGGGGCAGGTATGGG	1354
	ATGATGATGGGCAGCGC	1355
	GCGCTGCCCATCATCAT	1356
Melanoma Ser56Ile AGC-ATC	GCCGGCCCCCACCCTGGCTCTGACCATTCTGTTCTCTCTGGC AGGTCATGATGATGGGCAGCGCCCGAGTGGCGGAGCTGCTG CTGCTCCACGGCGCGGAGCCCAACTGCGCCGACCCCGC	1357
	GCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAGCAGCA GCAGCTCCGCCACTCGGGCGCTGCCCATCATCATGACCTGCC AGAGAGAACAGAATGGTCAGAGCCAGGGTGGGGGCCGGC	1358
	GATGGGCAGCGCCCGAG	1359
	CTCGGGCGCTGCCCATC	1360
Melanoma Ala57Val GCC-GTC	GGCCCCCACCCTGGCTCTGACCATTCTGTTCTCTCTGGCAGG TCATGATGATGGGCAGCGCCCGAGTGGCGGAGCTGCTGCTG CTCCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCAC	1361
	GTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAGCA GCAGCAGCTCCGCCACTCGGGCGCTGCCCATCATCATGACCT GCCAGAGAGAACAGAATGGTCAGAGCCAGGGTGGGGGCC	1362
	GGGCAGCGCCCGAGTGG	1363
	CCACTCGGGCGCTGCCC	1364
Melanoma Arg58Term cCGA-TGA	CCCCCACCCTGGCTCTGACCATTCTGTTCTCTCTGGCAGGTC ATGATGATGGGCAGCGCCCGAGTGGCGGAGCTGCTGCTGCT CCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCACTC	1365
	GAGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAG CAGCAGCAGCTCCGCCACTCGGGCGCTGCCCATCATCATGAC CTGCCAGAGAGAACAGAATGGTCAGAGCCAGGGTGGGGG	1366
	GCAGCGCCCGAGTGGCG	1367
	CGCCACTCGGGCGCTGC	1368
Melanoma Val59Gly GTG-GGG	CACCCTGGCTCTGACCATTCTGTTCTCTCTGGCAGGTCATGAT GATGGGCAGCGCCCGAGTGGCGGAGCTGCTGCTGCTCCACG GCGCGGAGCCCAACTGCGCCGACCCCGCCACTCTCAC	1369
	GTGAGAGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTG GAGCAGCAGCAGCTCCGCCACTCGGGCGCTGCCCATCATCA TGACCTGCCAGAGAGAACAGAATGGTCAGAGCCAGGGTG	1370
	CGCCCGAGTGGCGGAGC	1371

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCTCCGCCACTCGGGCG	1372
Melanoma Leu62Pro CTG-CCG	TCTGACCACTCTGCTCTCTCTGGCAGGTCATGATGATGGGCA GCGCCCGCGTGGCGGAGCTGCTGCTGCTCCACGGCGCGGA GCCCAACTGCGCAGACCCTGCCACTCTCACCCGACCGGT	1373
	ACCGGTCGGGTGAGAGTGGCAGGGTCTGCGCAGTTGGGCTC CGCGCCGTGGAGCAGCAGCAGCTCCGCCACGCGGGCGCTG CCCATCATCATGACCTGCCAGAGAGAGCAGAGTGGTCAGA	1374
	GGCGGAGCTGCTGCTGC	1375
	GCAGCAGCAGCTCCGCC	1376
Melanoma Ala68Val GCG-GTG	TCTGGCAGGTCATGATGATGGGCAGCGCCCGCGTGGCGGAG CTGCTGCTGCTCCACGGCGCGGAGCCCAACTGCGCAGACCC TGCCACTCTCACCCGACCGGTGCATGATGCTGCCCGGGA	1377
	TCCCGGGCAGCATCATGCACCGGTCGGGTGAGAGTGGCAGG GTCTGCGCAGTTGGGCTCCGCGCCGTGGAGCAGCAGCAGCT CCGCCACGCGGGCGCTGCCCATCATCATGACCTGCCAGA	1378
	CCACGGCGCGGAGCCCA	1379
	TGGGCTCCGCGCCGTGG	1380
Melanoma Asn71Lys AACt-AAA	CATGATGATGGGCAGCGCCCGAGTGGCGGAGCTGCTGCTGC TCCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCACTCTC ACCCGACCCGTGCACGACGCTGCCCGGGAGGGCTTCCTG	1381
	CAGGAAGCCCTCCCGGGCAGCGTCGTGCACGGGTCGGGTGA GAGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAG CAGCAGCAGCTCCGCCACTCGGGCGCTGCCCATCATCATG	1382
	GAGCCCAACTGCGCCGA	1383
	TCGGCGCAGTTGGGCTC	1384
Melanoma Asn71Ser AAC-AGC	TCATGATGATGGGCAGCGCCCGAGTGGCGGAGCTGCTGCTG CTCCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCACTCT CACCCGACCCGTGCACGACGCTGCCCGGGAGGGCTTCCT	1385
	AGGAAGCCCTCCCGGGCAGCGTCGTGCACGGGTCGGGTGAG AGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAGCA GCAGCAGCTCCGCCACTCGGGCGCTGCCCATCATCATGA	1386
	GGAGCCCAACTGCGCCG	1387
	CGGCGCAGTTGGGCTCC	1388
Melanoma Pro81Leu CCC-CTC	AGCTGCTGCTGCTCCACGGCGCGGAGCCCAACTGCGCCGAC CCCGCCACTCTCACCCGACCCGTGCACGACGCTGCCCGGGA GGGCTTCCTGGACACGCTGGTGGTGCTGCACCGGGCCGG	1389
	CCGGCCCGGTGCAGCACCACCAGCGTGTCCAGGAAGCCCTC CCCCGCAGCGTCGTGCACGGGTCGGGTGAGAGTGGCGGGG TCGGCGCAGTTGGGCTCCGCGCCGTGGAGCAGCAGCAGCT	1390
	CACCCGACCCGTGCACG	1391

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CGTGCACGGGTCGGGTG	1392
Melanoma Asp84Tyr cGAC-TAC	CTGCTCCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCAC TCTCACCCGACCCGTGCACGACGCTGCCCGGGAGGGCTTCC TGGACACGCTGGTGGTGCTGCACCGGGCCGGGGCGCGGC	1393
	GCCGCGCCCCGGCCCGGTGCAGCACCACCAGCGTGTCCAGG AAGCCCTCCCGGGCAGCGTCGTGCACGGGTCGGGTGAGAGT GGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAGCAG	1394
	CCGTGCACGACGCTGCC	1395
	GGCAGCGTCGTGCACGG	1396
Melanoma Ala85Thr cGCT-ACT	CTCCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCACTCT CACCCGACCCGTGCACGACGCTGCCCGGGAGGGCTTCCTGG ACACGCTGGTGGTGCTGCACCGGGCCGGGGCGCGGCTGG	1397
	CCAGCCGCGCCCCGGCCCGGTGCAGCACCACCAGCGTGTCC AGGAAGCCCTCCCGGGCAGCGTCGTGCACGGGTCGGGTGAG AGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAG	1398
	TGCACGACGCTGCCCGG	1399
	CCGGGCAGCGTCGTGCA	1400
Melanoma Arg87Pro CGG-CCG	GCGCGGAGCCCAACTGCGCCGACCCCGCCACTCTCACCCGA CCCGTGCACGACGCTGCCCGGGAGGGCTTCCTGGACACGCT GGTGGTGCTGCACCGGGCCGGGGCGCGGCTGGACGTGCG	1401
	CGCACGTCCAGCCGCGCCCCGGCCCGGTGCAGCACCACCAG CGTGTCCAGGAAGCCCTCCCGGGCAGCGTCGTGCACGGGTC GGGTGAGAGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGC	1402
	CGCTGCCCGGGAGGGCT	1403
	AGCCCTCCCGGGCAGCG	1404
Melanoma Arg87Trp cCGG-TGG	GGCGCGGAGCCCAACTGCGCCGACCCCGCCACTCTCACCCG ACCCGTGCACGACGCTGCCCGGGAGGGCTTCCTGGACACGC TGGTGGTGCTGCACCGGGCCGGGGCGCGGCTGGACGTGC	1405
	GCACGTCCAGCCGCGCCCCGGCCCGGTGCAGCACCACCAGC GTGTCCAGGAAGCCCTCCCGGGCAGCGTCGTGCACGGGTCG GGTGAGAGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCC	1406
	ACGCTGCCCGGGAGGGC	1407
	GCCCTCCCGGGCAGCGT	1408
Melanoma Leu97Arg CTG-CGG	CTCTCACCCGACCGGTGCATGATGCTGCCCGGGAGGGCTTC CTGGACACGCTGGTGGTGCTGCACCGGGCCGGGGCGCGGCT GGACGTGCGCGATGCCTGGGGTCGTCTGCCCGTGGACTT	1409
	AAGTCCACGGGCAGACGACCCCAGGCATCGCGCACGTCCAG CCGCGCCCCGGCCCGGTCGCAGCACCACCAGCGTGTCCAGGA AGCCCTCCCGGGCAGCATCATGCACCGGTCGGGTGAGAG	1410
	GGTGGTGCTGCACCGGG	1411

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCCGGTGCAGCACCACC	1412
Melanoma Arg99Pro CGG-CCG	CCCGACCGGTGCATGATGCTGCCCGGGAGGGCTTCCTGGAC ACGCTGGTGGTGCTGCACCGGGCCGGGGCGCGGCTGGACG TGCGCGATGCCTGGGGTCGTCTGCCCGTGGACTTGGCCGA	1413
	TCGGCCAAGTCCACGGGCAGACGACCCCAGGCATCGCGCAC GTCCAGCCGCGCCCCGGCCCGGTGCAGCACCACCAGCGTGT CCAGGAAGCCCTCCCGGGCAGCATCATGCACCGGTCGGG	1414
	GCTGCACCGGGCCGGGG	1415
	CCCCGGCCCGGTGCAGC	1416
Melanoma Gly101Trp cGGG-TGG	CCGGTGCATGATGCTGCCCGGGAGGGCTTCCTGGACACGCT GGTGGTGCTGCACCGGGCCGGGGCGCGGCTGGACGTGCGC GATGCCTGGGGTCGTCTGCCCGTGGACTTGGCCGAGGAGC	1417
	GCTCCTCGGCCAAGTCCACGGGCAGACGACCCCAGGCATCG CGCACGTCCAGCCGCGCCCCGGCCCGGTGCAGCACCACCAG CGTGTCCAGGAAGCCCTCCCGGGCAGCATCATGCACCGG	1418
	ACCGGGCCGGGGCGCGG	1419
	CCGCGCCCCGGCCCGGT	1420
Melanoma Arg107Cys gCGC-TGC	CGGGAGGGCTTCCTGGACACGCTGGTGGTGCTGCACCGGGC CGGGGCGCGGCTGGACGTGCGCGATGCCTGGGGTCGTCTGC CCGTGGACTTGGCCGAGGAGCGGGGCCACCGCGACGTTG	1421
	CAACGTCGCGGTGGCCCCGCTCCTCGGCCAAGTCCACGGGC AGACGACCCCAGGCATCGCGCACGTCCAGCCGCGCCCCGGC CCGGTGCAGCACCACCAGCGTGTCCAGGAAGCCCTCCCG	1422
	TGGACGTGCGCGATGCC	1423
	GGCATCGCGCACGTCCA	1424
Melanoma Ala118Thr gGCT-ACT	CACCGGGCCGGGGCGCGGCTGGACGTGCGCGATGCCTGGG GCCGTCTGCCCGTGGACCTGGCTGAGGAGCTGGGCCATCGC GATGTCGCACGGTACCTGCGCGCGGCTGCGGGGGGCACCA	1425
	TGGTGCCCCCCGCAGCCGCGCGCAGGTACCGTGCGACATCG CGATGGCCCAGCTCCTCAGCCAGGTCCACGGGCAGACGGCC CCAGGCATCGCGCACGTCCAGCCGCGCCCCGGCCCGGTG	1426
	TGGACCTGGCTGAGGAG	1427
	CTCCTCAGCCAGGTCCA	1428
Melanoma Val126Asp GTC-GAC	TGCGCGATGCCTGGGGCCGTCTGCCCGTGGACCTGGCTGAG GAGCTGGGCCATCGCGATGTCGCACGGTACCTGCGCGCGGC TGCGGGGGGCACCAGAGGCAGTAACCATGCCCGCATAGA	1429
	TCTATGCGGGCATGGTTACTGCCTCTGGTGCCCCCCGCAGCC GCGCGCAGGTACCGTGCGACATCGCGATGGCCCACGCCCTC AGCCAGGTCCACGGGCAGACGGCCCCAGGCATCGCGCA	1430
	TCGCGATGTCGCACGGT	1431

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACCGTGCGACATCGCGA	1432

EXAMPLE 11
Adenomatous polyposis of the colon - APC

Adenomatous polyposis of the colon is characterized by adenomatous polyps of the colon and rectum; in extreme cases the bowel is carpeted with a myriad of polyps. This is a viciously premalignant disease with one or more polyps progressing through dysplasia to malignancy in untreated gene carriers with a median age at diagnosis of 40 years.

Mutations in the APC gene are an initiating event for both familial and sporadic colorectal tumorigenesis and many alleles of the APC gene have been identified. Carcinoma may arise at any age from late childhood through the seventh decade with presenting features including, for example, weight loss and inanition, bowel obstruction, or bloody diarrhea. Cases of new mutation still present in these ways but in areas with well organized registers most other gene carriers are detected. The attached table discloses the correcting oligonucleotide base sequences for the APC oligonucleotides of the invention.

Table 18
APC Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Arg121Term AGA-TGA	GGATCTGTATCAAGCCGTTCTGGAGAGTGCAGTCCTGTTCCT ATGGGTTCATTTCCAAGAAGAGGGTTTGTAAATGGAAGCAGA GAAAGTACTGGATATTTAGAAGAACTTGAGAAAGAGA	1433
	TCTCTTTCTCAAGTTCTTCTAAATATCCAGTACTTTCTCTGCTT CCATTTACAAACCCTCTTCTTGGAAATGAACCCATAGGAACAG GACTGCACTCTCCAGAACGGCTTGATACAGATCC	1434
	TTCCAAGAAGAGGGTTT	1435
	AAACCCTCTTCTTGGAA	1436
Adenomatous polyposis coli Trp157Term TGG-TAG	AAAAAAAAAATAGGTCATTGCTTCTTGCTGATCTTGACAAAGAA GAAAAGGAAAAAGACTGGTATTACGCTCAACTTCAGAATCTCA CTAAAAGAATAGATAGTCTTCCTTTAACTGAAAA	1437
	TTTTCAGTTAAAGGAAGACTATCTATTCTTTTAGTGAGATTCTG AAGTTGAGCGTAATACCAGTCTTTTTCCTTTTCTTCTTTGTCAA GATCAGCAAGAAGCAATGACCTATTTTTTTTTT	1438
	AAAAGACTGGTATTACG	1439
	CGTAATACCAGTCTTTT	1440
Adenomatous polyposis coli Tyr159Term TAC-TAG	AAATAGGTCATTGCTTCTTGCTGATCTTGACAAAGAAGAAAAG GAAAAAGACTGGTATTACGCTCAACTTCAGAATCTCACTAAAA GAATAGATAGTCTTCCTTTAACTGAAAATGTAAGT	1441
	ACTTACATTTTCAGTTAAAGGAAGACTATCTATTCTTTTAGTGA GATTCTGAAGTTGAGCGTAATACCAGTCTTTTTCCTTTTCTTCT TTGTCAAGATCAGCAAGAAGCAATGACCTATTT	1442
	TGGTATTACGCTCAACT	1443
	AGTTGAGCGTAATACCA	1444
Adenomatous polyposis coli Gln163Term CAG-TAG	TTGCTTCTTGCTGATCTTGACAAAGAAGAAAAGGAAAAAGACT GGTATTACGCTCAACTTCAGAATCTCACTAAAAGAATAGATAG TCTTCCTTTAACTGAAAATGTAAGTAACTGGCAGT	1445
	ACTGCCAGTTACTTACATTTTCAGTTAAAGGAAGACTATCTATT CTTTTAGTGAGATTCTGAAGTTGAGCGTAATACCAGTCTTTTTC CTTTTCTTCTTTGTCAAGATCAGCAAGAAGCAA	1446
	CTCAACTTCAGAATCTC	1447
	GAGATTCTGAAGTTGAG	1448
Adenomatous polyposis coli Arg168Term AGA-TGA	CTTGACAAAGAAGAAAAGGAAAAAGACTGGTATTACGCTCAAC TTCAGAATCTCACTAAAAGAATAGATAGTCTTCCTTTAACTGAA AATGTAAGTAACTGGCAGTACAACTTATTTGAAA	1449
	TTTCAAATAAGTTGTACTGCCAGTTACTTACATTTTCAGTTAAA GGAAGACTATCTATTCTTTTAGTGAGATTCTGAAGTTGAGCGT AATACCAGTCTTTTTCCTTTTCTTCTTTGTCAAG	1450

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCACTAAAAGAATAGAT	1451
	ATCTATTCTTTTAGTGA	1452
Adenomatous polyposis coli Ser171Ile AGT-ATT	AAGAAAAGGAAAAAGACTGGTATTACGCTCAACTTCAGAATCT CACTAAAAGAATAGATAGTCTTCCTTTAACTGAAAATGTAAGTA ACTGGCAGTACAACTTATTTGAAACTTTAATAAC	1453
	GTTATTAAAGTTTCAAATAAGTTGTACTGCCAGTTACTTACATT TTCAGTTAAAGGAAGACTATCTATTCTTTTAGTGAGATTCTGAA GTTGAGCGTAATACCAGTCTTTTTCCTTTTCTT	1454
	AATAGATAGTCTTCCTT	1455
	AAGGAAGACTATCTATT	1456
Adenomatous polyposis coli Gln181Term CAA-TAA	GATTAACGTAAATACAAGATATTGATACTTTTTTATTATTTGTGG TTTTAGTTTTCCTTACAAACAGATATGACCAGAAGGCAATTGG AATATGAAGCAAGGCAAATCAGAGTTGCGATGG	1457
	CCATCGCAACTCTGATTTGCCTTGCTTCATATTCCAATTGCCT TCTGGTCATATCTGTTTGTAAGGAAAACTAAAACCACAAATAAT AAAAAAGTATCAATATCTTGTATTTACGTTAATC	1458
	TTTCCTTACAAACAGAT	1459
	ATCTGTTTGTAAGGAAA	1460
Adenomatous polyposis coli Glu190Term GAA-TAA	CTTTTTTATTATTTGTGGTTTTAGTTTTCCTTACAAACAGATATG ACCAGAAGGCAATTGGAATATGAAGCAAGGCAAATCAGAGTT GCGATGGAAGAACAACTAGGTACCTGCCAGGATA	1461
	TATCCTGGCAGGTACCTAGTTGTTCTTCCATCGCAACTCTGAT TTGCCTTGCTTCATATTCCAATTGCCTTCTGGTCATATCTGTTT GTAAGGAAAACTAAAACCACAAATAATAAAAAAG	1462
	GGCAATTGGAATATGAA	1463
	TTCATATTCCAATTGCC	1464
Adenomatous polyposis coli Gln208Term CAG-TAG	CAATTGGAATATGAAGCAAGGCAAATCAGAGTTGCGATGGAA GAACAACTAGGTACCTGCCAGGATATGGAAAAACGAGCACAG GTAAGTTACTTGTTTCTAAGTGATAAAACAGCGAAGA	1465
	TCTTCGCTGTTTTATCACTTAGAAACAAGTAACTTACCTGTGCT CGTTTTTCCATATCCTGGCAGGTACCTAGTTGTTCTTCCATCG CAACTCTGATTTGCCTTGCTTCATATTCCAATTG	1466
	GTACCTGCCAGGATATG	1467
	CATATCCTGGCAGGTAC	1468
Adenomatous polyposis coli Arg213Term CGA-TGA	GCAAGGCAAATCAGAGTTGCGATGGAAGAACAACTAGGTACC TGCCAGGATATGGAAAAACGAGCACAGGTAAGTTACTTGTTTC TAAGTGATAAAACAGCGAAGAGCTATTAGGAATAAA	1469
	TTTATTCCTAATAGCTCTTCGCTGTTTTATCACTTAGAAACAAG TAACTTACCTGTGCTCGTTTTTCCATATCCTGGCAGGTACCTA GTTGTTCTTCCATCGCAACTCTGATTTGCCTTGC	1470
	TGGAAAAACGAGCACAG	1471
	CTGTGCTCGTTTTTCCA	1472

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Arg232Term CGA-TGA	GTTTTATTTTAGCGAAGAATAGCCAGAATTCAGCAAATCGAAA AGGACATACTTCGTATA**C**GACAGCTTTTACAGTCCCAAGCAAC AGAAGCAGAGGTTAGTAAATTGCCTTTCTTGTTTG	1473
	CAAACAAGAAAGGCAATTTACTAACCTCTGCTTCTGTTGCTTG GGACTGTAAAAGCTGTC**G**TATACGAAGTATGTCCTTTTCGATT TGCTGAATTCTGGCTATTCTTCGCTAAAATAAAAC	1474
	TTCGTATA**C**GACAGCTT	1475
	AAGCTGTC**G**TATACGAA	1476
Adenomatous polyposis coli Gln233Term CAG-TAG	TTATTTTAGCGAAGAATAGCCAGAATTCAGCAAATCGAAAAGG ACATACTTCGTATACGA**C**AGCTTTTACAGTCCCAAGCAACAGA AGCAGAGGTTAGTAAATTGCCTTTCTTGTTTGTGG	1477
	CCACAAACAAGAAAGGCAATTTACTAACCTCTGCTTCTGTTGC TTGGGACTGTAAAAGCT**G**TCGTATACGAAGTATGTCCTTTTCG ATTTGCTGAATTCTGGCTATTCTTCGCTAAAATAA	1478
	GTATACGA**C**AGCTTTTA	1479
	TAAAAGCT**G**TCGTATAC	1480
Adenomatous polyposis coli Gln247Term CAG-TAG	AGAAAGCCTACACCATTTTTGCATGTACTGATGTTAACTCCAT CTTAACAGAGGTCATCT**C**AGAACAAGCATGAAACCGGCTCAC ATGATGCTGAGCGGCAGAATGAAGGTCAAGGAGTGG	1481
	CCACTCCTTGACCTTCATTCTGCCGCTCAGCATCATGTGAGC CGGTTTCATGCTTGTTCT**G**AGATGACCTCTGTTAAGATGGAGT TAACATCAGTACATGCAAAAATGGTGTAGGCTTTCT	1482
	GGTCATCT**C**AGAACAAG	1483
	CTTGTTCT**G**AGATGACC	1484
Adenomatous polyposis coli Gly267Term GGA-TGA	CAGAACAAGCATGAAACCGGCTCACATGATGCTGAGCGGCAG AATGAAGGTCAAGGAGTG**G**GAGAAATCAACATGGCAACTTCT GGTAATGGTCAGGTAAATAAATTATTTTATCATATTT	1485
	AAATATGATAAAATAATTTATTTACCTGACCATTACCAGAAGTT GCCATGTTGATTTCTC**C**CACTCCTTGACCTTCATTCTGCCGCT CAGCATCATGTGAGCCGGTTTCATGCTTGTTCTG	1486
	AAGGAGTG**G**GAGAAATC	1487
	GATTTCTC**C**CACTCCTT	1488
Adenomatous polyposis coli Glu443Term GAA-TAA	CTTCAAATAACAAAGCATTATGGTTTATGTTGATTTTATTTTTCA GTGCCAGCTCCTGTT**G**AACATCAGATCTGTCCTGCTGTGTGT GTTCTAATGAAACTTTCATTTGATGAAGAGCATA	1489
	TATGCTCTTCATCAAATGAAAGTTTCATTAGAACACACACAGCA GGACAGATCTGATGTT**C**AACAGGAGCTGGCACTGAAAAATAA AATCAACATAAACCATAATGCTTTGTTATTTGAAG	1490
	CTCCTGTT**G**AACATCAG	1491
	CTGATGTT**C**AACAGGAG	1492
Adenomatous polyposis coli SER457TER TCA-TAA	CAGTGCCAGCTCCTGTTGAACATCAGATCTGTCCTGCTGTGT GTGTTCTAATGAAACTTT**C**ATTTGATGAAGAGCATAGACATGC AATGAATGAACTAGGTAAGACAAAAATGTTTTTTAA	1493

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTAAAAAACATTTTTGTCTTACCTAGTTCATTCATTGCATGTCTA TGCTCTTCATCAAATGAAAGTTTCATTAGAACACACACAGCAG GACAGATCTGATGTTCAACAGGAGCTGGCACTG	1494
	GAAACTTTCATTTGATG	1495
	CATCAAATGAAAGTTTC	1496
Adenomatous polyposis coli Gln473Term CAG-TAG	AGTTGTTTTATTTTAGATGATTGTCTTTTTCCTCTTGCCCTTTTT AAATTAGGGGGACTACAGGCCATTGCAGAATTATTGCAAGTG GACTGTGAAATGTACGGGCTTACTAATGACCACT	1497
	AGTGGTCATTAGTAAGCCCGTACATTTCACAGTCCACTTGCAA TAATTCTGCAATGGCCTGTAGTCCCCCTAATTTAAAAAGGGCA AGAGGAAAAAGACAATCATCTAAAATAAAACAACT	1498
	GGGGACTACAGGCCATT	1499
	AATGGCCTGTAGTCCCC	1500
Adenomatous polyposis coli Tyr486Term TAC-TAG	TTTTAAATTAGGGGGACTACAGGCCATTGCAGAATTATTGCAA GTGGACTGTGAAATGTACGGGCTTACTAATGACCACTACAGTA TTACACTAAGACGATATGCTGGAATGGCTTTGACA	1501
	TGTCAAAGCCATTCCAGCATATCGTCTTAGTGTAATACTGTAG TGGTCATTAGTAAGCCCGTACATTTCACAGTCCACTTGCAATA ATTCTGCAATGGCCTGTAGTCCCCCTAATTTAAAA	1502
	GAAATGTACGGGCTTAC	1503
	GTAAGCCCGTACATTTC	1504
Adenomatous polyposis coli Arg499Term CGA-TGA	TTGCAAGTGGACTGTGAAATGTATGGGCTTACTAATGACCACT ACAGTATTACACTAAGACGATATGCTGGAATGGCTTTGACAAA CTTGACTTTTGGAGATGTAGCCAACAAGGTATGTT	1505
	AACATACCTTGTTGGCTACATCTCCAAAAGTCAAGTTTGTCAA AGCCATTCCAGCATATCGTCTTAGTGTAATACTGTAGTGGTCA TTAGTAAGCCCATACATTTCACAGTCCACTTGCAA	1506
	CACTAAGACGATATGCT	1507
	AGCATATCGTCTTAGTG	1508
Adenomatous polyposis coli Tyr500Term TAT-TAG	AGTGGACTGTGAAATGTATGGGCTTACTAATGACCACTACAGT ATTACACTAAGACGATATTGCTGGAATGGCTTTGACAAACTTGA CTTTTGGAGATGTAGCCAACAAGGTATGTTTTTAT	1509
	ATAAAAACATACCTTGTTGGCTACATCTCCAAAAGTCAAGTTTG TCAAAGCCATTCCAGCATATCGTCTTAGTGTAATACTGTAGTG GTCATTAGTAAGCCCATACATTTCACAGTCCACT	1510
	AGACGATATTGCTGGAAT	1511
	ATTCCAGCATATCGTCT	1512
Adenomatous polyposis coli Lys586Term AAA-TAA	GACAAATTCCAACTCTAATTAGATGACCCATATTCTGTTTCTTA CTAGGAATCAACCCTCAAAAGCGTATTGAGTGCCTTATGGAAT TTGTCAGCACATTGCACTGAGAATAAAGCTGATA	1513
	TATCAGCTTTATTCTCAGTGCAATGTGCTGACAAATTCCATAA GGCACTCAATACGCTTTTGAGGGTTGATTCCTAGTAAGAAACA GAATATGGGTCATCTAATTAGAGTTGGAATTTGTC	1514
	CAACCCTCAAAAGCGTA	1515

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TACGCTTTTGAGGGTTG	1516
Adenomatous polyposis coli Leu592Term TTA-TGA	TAGATGACCCATATTCTGTTTCTTACTAGGAATCAACCCTCAAA AGCGTATTGAGTGCCTTATGGAATTTGTCAGCACATTGCACTG AGAATAAAGCTGATATATGTGCTGTAGATGGTGC	1517
	GCACCATCTACAGCACATATATCAGCTTTATTCTCAGTGCAAT GTGCTGACAAATTCCATAAGGCACTCAATACGCTTTTGAGGGT TGATTCCTAGTAAGAAACAGAATATGGGTCATCTA	1518
	GAGTGCCTTATGGAATT	1519
	AATTCCATAAGGCACTC	1520
Adenomatous polyposis coli Trp593Term TGG-TAG	ATGACCCATATTCTGTTTCTTACTAGGAATCAACCCTCAAAAG CGTATTGAGTGCCTTATGGAATTTGTCAGCACATTGCACTGAG AATAAAGCTGATATATGTGCTGTAGATGGTGCACT	1521
	AGTGCACCATCTACAGCACATATATCAGCTTTATTCTCAGTGC AATGTGCTGACAAATTCCATAAGGCACTCAATACGCTTTTGAG GGTTGATTCCTAGTAAGAAACAGAATATGGGTCAT	1522
	TGCCTTATGGAATTTGT	1523
	ACAAATTCCATAAGGCA	1524
Adenomatous polyposis coli Trp593Term TGG-TGA	TGACCCATATTCTGTTTCTTACTAGGAATCAACCCTCAAAAGC GTATTGAGTGCCTTATGGAATTTGTCAGCACATTGCACTGAGA ATAAAGCTGATATATGTGCTGTAGATGGTGCACTT	1525
	AAGTGCACCATCTACAGCACATATATCAGCTTTATTCTCAGTG CAATGTGCTGACAAATTCCATAAGGCACTCAATACGCTTTTGA GGGTTGATTCCTAGTAAGAAACAGAATATGGGTCA	1526
	GCCTTATGGAATTTGTC	1527
	GACAAATTCCATAAGGC	1528
Adenomatous polyposis coli Tyr622Term TAC-TAA	TAAAGCTGATATATGTGCTGTAGATGGTGCACTTGCATTTTTG GTTGGCACTCTTACTTACCGGAGCCAGACAAACACTTTAGCC ATTATTGAAAGTGGAGGTGGGATATTACGGAATGTG	1529
	CACATTCCGTAATATCCCACCTCCACTTTCAATAATGGCTAAA GTGTTTGTCTGGCTCCGGTAAGTAAGAGTGCCAACCAAAAAT GCAAGTGCACCATCTACAGCACATATATCAGCTTTA	1530
	CTTACTTACCGGAGCCA	1531
	TGGCTCCGGTAAGTAAG	1532
Adenomatous polyposis coli Gln625Term CAG-TAG	GATATATGTGCTGTAGATGGTGCACTTGCATTTTTGGTTGGCA CTCTTACTTACCGGAGCCAGACAAACACTTTAGCCATTATTGA AAGTGGAGGTGGGATATTACGGAATGTGTCCAGCT	1533
	AGCTGGACACATTCCGTAATATCCCACCTCCACTTTCAATAAT GGCTAAAGTGTTTGTCTGGCTCCGGTAAGTAAGAGTGCCAAC CAAAAATGCAAGTGCACCATCTACAGCACATATATC	1534
	ACCGGAGCCAGACAAAC	1535
	GTTTGTCTGGCTCCGGT	1536

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Leu629Term TTA-TAA	TAGATGGTGCACTTGCATTTTTGGTTGGCACTCTTACTTACCG GAGCCAGACAAACACTTTAGCCATTATTGAAAGTGGAGGTGG GATATTACGGAATGTGTCCAGCTTGATAGCTACAAA	1537
	TTTGTAGCTATCAAGCTGGACACATTCCGTAATATCCCACCTC CACTTTCAATAATGGCTAAAGTGTTTGTCTGGCTCCGGTAAGT AAGAGTGCCAACCAAAAATGCAAGTGCACCATCTA	1538
	AAACACTTTAGCCATTA	1539
	TAATGGCTAAAGTGTTT	1540
Adenomatous polyposis coli Glu650Term GAG-TAG	GCCATTATTGAAAGTGGAGGTGGGATATTACGGAATGTGTCC AGCTTGATAGCTACAAATGAGGACCACAGGTATATATAGAGTT TTATATTACTTTTAAAGTACAGAATTCATACTCTCA	1541
	TGAGAGTATGAATTCTGTACTTTAAAAGTAATATAAAACTCTAT ATATACCTGTGGTCCTCATTTGTAGCTATCAAGCTGGACACAT TCCGTAATATCCCACCTCCACTTTCAATAATGGC	1542
	CTACAAATGAGGACCAC	1543
	GTGGTCCTCATTTGTAG	1544
Adenomatous polyposis coli Trp699Term TGG-TGA	TGCATGTGGAACTTTGTGGAATCTCTCAGCAAGAAATCCTAAA GACCAGGAAGCATTATGGGACATGGGGGCAGTTAGCATGCTC AAGAACCTCATTCATTCAAAGCACAAAATGATTGCT	1545
	AGCAATCATTTTGTGCTTTGAATGAATGAGGTTCTTGAGCATG CTAACTGCCCCCATGTCCCATAATGCTTCCTGGTCTTTAGGAT TTCTTGCTGAGAGATTCCACAAAGTTCCACATGCA	1546
	GCATTATGGGACATGGG	1547
	CCCATGTCCCATAATGC	1548
Adenomatous polyposis coli Ser713Term TCA-TGA	AAGACCAGGAAGCATTATGGGACATGGGGGCAGTTAGCATGC TCAAGAACCTCATTCATTCAAAGCACAAAATGATTGCTATGGG AAGTGCTGCAGCTTTAAGGAATCTCATGGCAAATAG	1549
	CTATTTGCCATGAGATTCCTTAAAGCTGCAGCACTTCCCATAG CAATCATTTTGTGCTTTGAATGAATGAGGTTCTTGAGCATGCT AACTGCCCCCATGTCCCATAATGCTTCCTGGTCTT	1550
	CATTCATTCAAAGCACA	1551
	TGTGCTTTGAATGAATG	1552
Adenomatous polyposis coli Ser722Gly AGT-GGT	GGGGCAGTTAGCATGCTCAAGAACCTCATTCATTCAAAGCAC AAAATGATTGCTATGGGAAGTGCTGCAGCTTTAAGGAATCTCA TGGCAAATAGGCCTGCGAAGTACAAGGATGCCAATA	1553
	TATTGGCATCCTTGTACTTCGCAGGCCTATTTGCCATGAGATT CCTTAAAGCTGCAGCACTTCCCATAGCAATCATTTTGTGCTTT GAATGAATGAGGTTCTTGAGCATGCTAACTGCCCC	1554
	CTATGGGAAGTGCTGCA	1555
	TGCAGCACTTCCCATAG	1556

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Leu764Term TTA-TAA	TCTCCTGGCTCAGCTTGCCATCTCTTCATGTTAGGAAACAAAA AGCCCTAGAAGCAGAATTAGATGCTCAGCACTTATCAGAAACT TTTGACAATATAGACAATTTAAGTCCCAAGGCATC	1557
	GATGCCTTGGGACTTAAATTGTCTATATTGTCAAAAGTTTCTGA TAAGTGCTGAGCATCTAATTCTGCTTCTAGGGCTTTTTGTTTC CTAACATGAAGAGATGGCAAGCTGAGCCAGGAGA	1558
	AGCAGAATTAGATGCTC	1559
	GAGCATCTAATTCTGCT	1560
Adenomatous polyposis coli Ser784Thr TCT-ACT	TTAGATGCTCAGCACTTATCAGAAACTTTTGACAATATAGACAA TTTAAGTCCCAAGGCATCTCATCGTAGTAAGCAGAGACACAG CAAGTCTCTATGGTGATTATGTTTTTGACACCATC	1561
	GATGGTGTCAAAAACATAATCACCATAGAGACTTGCTGTGTCT CTGCTTACTACGATGAGATGCCTTGGGACTTAAATTGTCTATA TTGTCAAAAGTTTCTGATAAGTGCTGAGCATCTAA	1562
	CCAAGGCATCTCATCGT	1563
	ACGATGAGATGCCTTGG	1564
Adenomatous polyposis coli Arg805Term CGA-TGA	CTCATCGTAGTAAGCAGAGACACAGCAAGTCTCTATGGTGATT ATGTTTTTGACACCAATCGACATGATGATAATAGGTCAGACAT TTTAATACTGGCACATGACTGTCCTTTCACCATAT	1565
	ATATGGTGAAAGGACAGTCATGTGCCAGTATTAAAATGTCTGA CCTATTATCATCATGTCGATTGGTGTCAAAAACATAATCACCAT AGAGACTTGCTGTGTCTCTGCTTACTACGATGAG	1566
	ACACCAATCGACATGAT	1567
	ATCATGTCGATTGGTGT	1568
Adenomatous polyposis coli Gln879Term CAG-TAG	GGTCTAGGCAACTACCATCCAGCAACAGAAAATCCAGGAACT TCTTCAAAGCGAGGTTTGCAGATCTCCACCACTGCAGCCCAG ATTGCCAAAGTCATGGAAGAAGTGTCAGCCATTCATA	1569
	TATGAATGGCTGACACTTCTTCCATGACTTTGGCAATCTGGGC TGCAGTGGTGGAGATCTGCAAACCTCGCTTTGAAGAAGTTCC TGGATTTTCTGTTGCTGGATGGTAGTTGCCTAGACC	1570
	GAGGTTTGCAGATCTCC	1571
	GGAGATCTGCAAACCTC	1572
Adenomatous polyposis coli Ser932Term TCA-TAA	TACATTGTGTGACAGATGAGAGAAATGCACTTAGAAGAAGCTC TGCTGCCCATACACATTCAAACACTTACAATTTCACTAAGTCG GAAAATTCAAATAGGACATGTTCTATGCCTTATGC	1573
	GCATAAGGCATAGAACATGTCCTATTTGAATTTTCCGACTTAG TGAAATTGTAAGTGTTTGAATGTGTATGGGCAGCAGAGCTTCT TCTAAGTGCATTTCTCTCATCTGTCACACAATGTA	1574
	TACACATTCAAACACTT	1575
	AAGTGTTTGAATGTGTA	1576
Adenomatous polyposis coli Ser932Term TCA-TGA	TACATTGTGTGACAGATGAGAGAAATGCACTTAGAAGAAGCTC TGCTGCCCATACACATTCAAACACTTACAATTTCACTAAGTCG GAAAATTCAAATAGGACATGTTCTATGCCTTATGC	1577

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCATAAGGCATAGAACATGTCCTATTTGAATTTTCCGACTTAG TGAAATTGTAAGTGTTTGAATGTGTATGGGCAGCAGAGCTTCT TCTAAGTGCATTTCTCTCATCTGTCACACAATGTA	1578
	TACACATTCAAACACTT	1579
	AAGTGTTTGAATGTGTA	1580
Adenomatous polyposis coli Tyr935Term TAC-TAG	GACAGATGAGAGAAATGCACTTAGAAGAAGCTCTGCTGCCCA TACACATTCAAACACTTACAATTTCACTAAGTCGGAAAATTCAA ATAGGACATGTTCTATGCCTTATGCCAAATTAGAA	1581
	TTCTAATTTGGCATAAGGCATAGAACATGTCCTATTTGAATTTT CCGACTTAGTGAAATTGTAAGTGTTTGAATGTGTATGGGCAGC AGAGCTTCTTCTAAGTGCATTTCTCTCATCTGTC	1582
	AACACTTACAATTTCAC	1583
	GTGAAATTGTAAGTGTT	1584
Adenomatous polyposis coli Tyr935Term TAC-TAA	GACAGATGAGAGAAATGCACTTAGAAGAAGCTCTGCTGCCCA TACACATTCAAACACTTACAATTTCACTAAGTCGGAAAATTCAA ATAGGACATGTTCTATGCCTTATGCCAAATTAGAA	1585
	TTCTAATTTGGCATAAGGCATAGAACATGTCCTATTTGAATTTT CCGACTTAGTGAAATTGTAAGTGTTTGAATGTGTATGGGCAGC AGAGCTTCTTCTAAGTGCATTTCTCTCATCTGTC	1586
	AACACTTACAATTTCAC	1587
	GTGAAATTGTAAGTGTT	1588
Adenomatous polyposis coli Tyr1000Term TAC-TAA	ACCCTCGATTGAATCCTATTCTGAAGATGATGAAAGTAAGTTTT GCAGTTATGGTCAATACCCAGCCGACCTAGCCCATAAAATACA TAGTGCAAATCATATGGATGATAATGATGGAGAA	1589
	TTCTCCATCATTATCATCCATATGATTTGCACTATGTATTTTAT GGGCTAGGTCGGCTGGGTATTGACCATAACTGCAAAACTTAC TTTCATCATCTTCAGAATAGGATTCAATCGAGGGT	1590
	GGTCAATACCCAGCCGA	1591
	TCGGCTGGGTATTGACC	1592
Adenomatous polyposis coli Glu1020Term GAA-TAA	TACCCAGCCGACCTAGCCCATAAAATACATAGTGCAAATCATA TGGATGATAATGATGGAGAACTAGATACACCAATAAATTATAG TCTTAAATATTCAGATGAGCAGTTGAACTCTGGAA	1593
	TTCCAGAGTTCAACTGCTCATCTGAATATTTAAGACTATAATTT ATTGGTGTATCTAGTTCTCCATCATTATCATCCATATGATTTGC ACTATGTATTTTATGGGCTAGGTCGGCTGGGTA	1594
	ATGATGGAGAACTAGAT	1595
	ATCTAGTTCTCCATCAT	1596
Adenomatous polyposis coli Ser1032Term TCA-TAA	ATGAAACCCTCGATTGAATCCTATTCTGAAGATGATGAAAGTA AGTTTTGCAGTTATGGTCAATACCCAGCCGACCTAGCCCATAA AATACATAGTGCAAATCATATGGATGATAATGATG	1597
	CATCATTATCATCCATATGATTTGCACTATGTATTTTATGGGCT AGGTCGGCTGGGTATTGACCATAACTGCAAAACTTACTTTCAT CATCTTCAGAATAGGATTCAATCGAGGGTTTCAT	1598

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GTTATGGTCAATACCCA	1599
	TGGGTATTGACCATAAC	1600
Adenomatous polyposis coli Gln1041Term CAA-TAA	TGAAGATGATGAAAGTAAGTTTTGCAGTTATGGTCAATACCCA GCCGACCTAGCCCATAAAATACATAGTGCAAATCATATGGATG ATAATGATGGAGAACTAGATACACCAATAAATTAT	1601
	ATAATTTATTGGTGTATCTAGTTCTCCATCATTATCATCCATAT GATTTGCACTATGTATTTTATGGGCTAGGTCGGCTGGGTATTG ACCATAACTGCAAAACTTACTTTCATCATCTTCA	1602
	GCCCATAAAATACATAG	1603
	CTATGTATTTTATGGGC	1604
Adenomatous polyposis coli Gln1045Term CAG-TAG	ATAAATTATAGTCTTAAATATTCAGATGAGCAGTTGAACTCTGG AAGGCAAAGTCCTTCACAGAATGAAAGATGGGCAAGACCCAA ACACATAATAGAAGATGAAATAAAACAAAGTGAGC	1605
	GCTCACTTTGTTTTATTTCATCTTCTATTATGTGTTTGGGTCTT GCCCATCTTTCATTCTGTGAAGGACTTTGCCTTCCAGAGTTCA ACTGCTCATCTGAATATTTAAGACTATAATTTAT	1606
	GTCCTTCACAGAATGAA	1607
	TTCATTCTGTGAAGGAC	1608
Adenomatous polyposis coli Gln1067Term CAA-TAA	GAAAGATGGGCAAGACCCAAACACATAATAGAAGATGAAATAA AACAAAGTGAGCAAAGACAATCAAGGAATCAAAGTACAACTTA TCCTGTTTATACTGAGAGCACTGATGATAAACACC	1609
	GGTGTTTATCATCAGTGCTCTCAGTATAAACAGGATAAGTTGT ACTTTGATTCCTTGATTGTCTTTGCTCACTTTGTTTTATTTCATC TTCTATTATGTGTTTGGGTCTTGCCCATCTTTC	1610
	AGCAAAGACAATCAAGG	1611
	CCTTGATTGTCTTTGCT	1612
Adenomatous polyposis coli Tyr1075Term TAT-TAG	AATAGAAGATGAAATAAAACAAAGTGAGCAAAGACAATCAAGG AATCAAAGTACAACTTATCCTGTTTATACTGAGAGCACTGATG ATAAACACCTCAAGTTCCAACCACATTTTGGACAG	1613
	CTGTCCAAAATGTGGTTGGAACTTGAGGTGTTTATCATCAGTG CTCTCAGTATAAACAGGATAAGTTGTACTTTGATTCCTTGATTG TCTTTGCTCACTTTGTTTTATTTCATCTTCTATT	1614
	ACAACTTATCCTGTTTA	1615
	TAAACAGGATAAGTTGT	1616
Adenomatous polyposis coli Tyr1102Term TAC-TAG	TGATGATAAACACCTCAAGTTCCAACCACATTTTGGACAGCAG GAATGTGTTTCTCCATACAGGTCACGGGGAGCCAATGGTTCA GAAACAAATCGAGTGGGTTCTAATCATGGAATTAAT	1617
	ATTAATTCCATGATTAGAACCCACTCGATTTGTTTCTGAACCAT TGGCTCCCCGTGACCTGTATGGAGAAACACATTCCTGCTGTC CAAAATGTGGTTGGAACTTGAGGTGTTTATCATCA	1618
	TCTCCATACAGGTCACG	1619
	CGTGACCTGTATGGAGA	1620

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Ser1110Term TCA-TGA	AACCACATTTTGGACAGCAGGAATGTGTTTCTCCATACAGGTC ACGGGGAGCCAATGGTTCAGAAACAAATCGAGTGGGTTCTAA TCATGGAATTAATCAAAATGTAAGCCAGTCTTTGTG	1621
	CACAAAGACTGGCTTACATTTTGATTAATTCCATGATTAGAACC CACTCGATTTGTTTCTGAACCATTGGCTCCCCGTGACCTGTAT GGAGAAACACATTCCTGCTGTCCAAAATGTGGTT	1622
	CAATGGTTCAGAAACAA	1623
	TTGTTTCTGAACCATTG	1624
Adenomatous polyposis coli Arg1114Term CGA-TGA	GGACAGCAGGAATGTGTTTCTCCATACAGGTCACGGGGAGCC AATGGTTCAGAAACAAATCGAGTGGGTTCTAATCATGGAATTA ATCAAAATGTAAGCCAGTCTTTGTGTCAAGAAGATG	1625
	CATCTTCTTGACACAAAGACTGGCTTACATTTTGATTAATTCCA TGATTAGAACCCACTCGATTTGTTTCTGAACCATTGGCTCCCC GTGACCTGTATGGAGAAACACATTCCTGCTGTCC	1626
	AAACAAATCGAGTGGGT	1627
	ACCCACTCGATTTGTTT	1628
Adenomatous polyposis coli Tyr1135Term TAT-TAG	GGGTTCTAATCATGGAATTAATCAAAATGTAAGCCAGTCTTTG TGTCAAGAAGATGACTATGAAGATGATAAGCCTACCAATTATA GTGAACGTTACTCTGAAGAAGAACAGCATGAAGAA	1629
	TTCTTCATGCTGTTCTTCTTCAGAGTAACGTTCACTATAATTGG TAGGCTTATCATCTTCATAGTCATCTTCTTGACACAAAGACTG GCTTACATTTTGATTAATTCCATGATTAGAACCC	1630
	GATGACTATGAAGATGA	1631
	TCATCTTCATAGTCATC	1632
Adenomatous polyposis coli Gln1152Term CAG-TAG	GAAGATGACTATGAAGATGATAAGCCTACCAATTATAGTGAAC GTTACTCTGAAGAAGAACAGCATGAAGAAGAAGAGAGACCAA CAAATTATAGCATAAAATATAATGAAGAGAAACGTC	1633
	GACGTTTCTCTTCATTATATTTTATGCTATAATTTGTTGGTCTCT CTTCTTCTTCATGCTGTTCTTCTTCAGAGTAACGTTCACTATAA TTGGTAGGCTTATCATCTTCATAGTCATCTTC	1634
	AAGAAGAACAGCATGAA	1635
	TTCATGCTGTTCTTCTT	1636
Adenomatous polyposis coli Gln1175Term CAG-TAG	GAAGAAGAGAGACCAACAAATTATAGCATAAAATATAATGAAG AGAAACGTCATGTGGATCAGCCTATTGATTATAGTTTAAAATAT GCCACAGATATTCCTTCATCACAGAAACAGTCAT	1637
	ATGACTGTTTCTGTGATGAAGGAATATCTGTGGCATATTTTAAA CTATAATCAATAGGCTGATCCACATGACGTTTCTCTTCATTATA TTTTATGCTATAATTTGTTGGTCTCTCTTCTTC	1638
	ATGTGGATCAGCCTATT	1639
	AATAGGCTGATCCACAT	1640

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Pro1176Leu CCT-CTT	AAGAGAGACCAACAAATTATAGCATAAAATATAATGAAGAGAA ACGTCATGTGGATCAGCCTATTGATTATAGTTTAAAATATGCCA CAGATATTCCTTCATCACAGAAACAGTCATTTTC	1641
	GAAAATGACTGTTTCTGTGATGAAGGAATATCTGTGGCATATT TTAAACTATAATCAATAGGCTGATCCACATGACGTTTCTCTTCA TTATATTTTATGCTATAATTTGTTGGTCTCTCTT	1642
	GGATCAGCCTATTGATT	1643
	AATCAATAGGCTGATCC	1644
Adenomatous polyposis coli Ala1184Pro GCC-CCC	ATAAAATATAATGAAGAGAAACGTCATGTGGATCAGCCTATTG ATTATAGTTTAAAATATGCCACAGATATTCCTTCATCACAGAAA CAGTCATTTTCATTCTCAAAGAGTTCATCTGGAC	1645
	GTCCAGATGAACTCTTTGAGAATGAAAATGACTGTTTCTGTGA TGAAGGAATATCTGTGGCATATTTTAAACTATAATCAATAGGCT GATCCACATGACGTTTCTCTTCATTATATTTTAT	1646
	TAAAATATGCCACAGAT	1647
	ATCTGTGGCATATTTTA	1648
Adenomatous polyposis coli Ser1194Term TCA-TGA	ATCAGCCTATTGATTATAGTTTAAAATATGCCACAGATATTCCT TCATCACAGAAACAGTCATTTTCATTCTCAAAGAGTTCATCTG GACAAAGCAGTAAAACCGAACATATGTCTTCAAG	1649
	CTTGAAGACATATGTTCGGTTTTACTGCTTTGTCCAGATGAAC TCTTTGAGAATGAAAATGACTGTTTCTGTGATGAAGGAATATCT GTGGCATATTTTAAACTATAATCAATAGGCTGAT	1650
	GAAACAGTCATTTTCAT	1651
	ATGAAAATGACTGTTTC	1652
Adenomatous polyposis coli Ser1198Term TCA-TGA	ATTATAGTTTAAAATATGCCACAGATATTCCTTCATCACAGAAA CAGTCATTTTCATTCTCAAAGAGTTCATCTGGACAAAGCAGTA AAACCGAACATATGTCTTCAAGCAGTGAGAATAC	1653
	GTATTCTCACTGCTTGAAGACATATGTTCGGTTTTACTGCTTTG TCCAGATGAACTCTTTGAGAATGAAAATGACTGTTTCTGTGAT GAAGGAATATCTGTGGCATATTTTAAACTATAAT	1654
	TTCATTCTCAAAGAGTT	1655
	AACTCTTTGAGAATGAA	1656
Adenomatous polyposis coli Gln1228Term CAG-TAG	ACCGAACATATGTCTTCAAGCAGTGAGAATACGTCCACACCTT CATCTAATGCCAAGAGGCAGAATCAGCTCCATCCAGTTCTGC ACAGAGTAGAAGTGGTCAGCCTCAAAGGCTGCCACT	1657
	AGTGGCAGCCTTTGAGGCTGACCACTTCTACTCTGTGCAGAA CTGGATGGAGCTGATTCTGCCTCTTGGCATTAGATGAAGGTG TGGACGTATTCTCACTGCTTGAAGACATATGTTCGGT	1658
	CCAAGAGGCAGAATCAG	1659
	CTGATTCTGCCTCTTGG	1660

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Gln1230Term CAG-TAG	CATATGTCTTCAAGCAGTGAGAATACGTCCACACCTTCATCTA ATGCCAAGAGGCAGAATCAGCTCCATCCAGTTCTGCACAGAG TAGAAGTGGTCAGCCTCAAAGGCTGCCACTTGCAAG	1661
	CTTGCAAGTGGCAGCCTTTGAGGCTGACCACTTCTACTCTGT GCAGAACTGGATGGAGCTGATTCTGCCTCTTGGCATTAGATG AAGGTGTGGACGTATTCTCACTGCTTGAAGACATATG	1662
	GGCAGAATCAGCTCCAT	1663
	ATGGAGCTGATTCTGCC	1664
Adenomatous polyposis coli Cys1249Term TGC-TGA	TCAGCTCCATCCAAGTTCTGCACAGAGTAGAAGTGGTCAGCC TCAAAAGGCTGCCACTTGCAAAGTTTCTTCTATTAACCAAGAA ACAATACAGACTTATTGTGTAGAAGATACTCCAATA	1665
	TATTGGAGTATCTTCTACACAATAAGTCTGTATTGTTTCTTGGT TAATAGAAGAAACTTTGCAAGTGGCAGCCTTTTGAGGCTGACC ACTTCTACTCTGTGCAGAACTTGGATGGAGCTGA	1666
	GCCACTTGCAAAGTTTC	1667
	GAAACTTTGCAAGTGGC	1668
Adenomatous polyposis coli Cys1270Term TGT-TGA	AGTTTCTTCTATTAACCAAGAAACAATACAGACTTATTGTGTAG AAGATACTCCAATATGTTTTTCAAGATGTAGTTCATTATCATCT TTGTCATCAGCTGAAGATGAAATAGGATGTAAT	1669
	ATTACATCCTATTTCATCTTCAGCTGATGACAAAGATGATAATG AACTACATCTTGAAAAACATATTGGAGTATCTTCTACACAATAA GTCTGTATTGTTTCTTGGTTAATAGAAGAAACT	1670
	CCAATATGTTTTTCAAG	1671
	CTTGAAAAACATATTGG	1672
Adenomatous polyposis coli Ser1276Term TCA-TGA	AAGAAACAATACAGACTTATTGTGTAGAAGATACTCCAATATGT TTTTCAAGATGTAGTTCATTATCATCTTTGTCATCAGCTGAAGA TGAAATAGGATGTAATCAGACGACACAGGAAGC	1673
	GCTTCCTGTGTCGTCTGATTACATCCTATTTCATCTTCAGCTG ATGACAAAGATGATAATGAACTACATCTTGAAAAACATATTGGA GTATCTTCTACACAATAAGTCTGTATTGTTTCTT	1674
	ATGTAGTTCATTATCAT	1675
	ATGATAATGAACTACAT	1676
Adenomatous polyposis coli Glu1286Term GAA-TAA	GATACTCCAATATGTTTTTCAAGATGTAGTTCATTATCATCTTT GTCATCAGCTGAAGATGAAATAGGATGTAATCAGACGACACA GGAAGCAGATTCTGCTAATACCCTGCAAATAGCAG	1677
	CTGCTATTTGCAGGGTATTAGCAGAATCTGCTTCCTGTGTCGT CTGATTACATCCTATTTCATCTTCAGCTGATGACAAAGATGATA ATGAACTACATCTTGAAAAACATATTGGAGTATC	1678
	CTGAAGATGAAATAGGA	1679
	TCCTATTTCATCTTCAG	1680

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Gln1294Term CAG-TAG	TGTAGTTCATTATCATCTTTGTCATCAGCTGAAGATGAAATAGG ATGTAATCAGACGACA**C**AGGAAGCAGATTCTGCTAATACCCTG CAAATAGCAGAAATAAAAGAAAAGATTGGAACTA	1681
	TAGTTCCAATCTTTTCTTTTATTTCTGCTATTTGCAGGGTATTA GCAGAATCTGCTTCCT**G**TGTCGTCTGATTACATCCTATTTCAT CTTCAGCTGATGACAAAGATGATAATGAACTACA	1682
	AGACGACA**C**AGGAAGCA	1683
	TGCTTCCT**G**TGTCGTCT	1684
Predisposition to, association with, colorectal cancer Ile1307Lys ATA-AAA	TAGGATGTAATCAGACGACACAGGAAGCAGATTCTGCTAATAC CCTGCAAATAGCAGAAA**T**AAAAGAAAAGATTGGAACTAGGTCA GCTGAAGATCCTGTGAGCGAAGTTCCAGCAGTGTC	1685
	GACACTGCTGGAACTTCGCTCACAGGATCTTCAGCTGACCTA GTTCCAATCTTTTCTTTT**A**TTTCTGCTATTTGCAGGGTATTAGC AGAATCTGCTTCCTGTGTCGTCTGATTACATCCTA	1686
	AGCAGAAA**T**AAAAGAAA	1687
	TTTCTTTT**A**TTTCTGCT	1688
Adenomatous polyposis coli Glu1309Term GAA-TAA	CCAAGAAACAATACAGACTTATTGTGTAGAAGATACTCCAATA TGTTTTTCAAGATGTAG**T**TCATTATCATCTTTGTCATCAGCTGA AGATGAAATAGGATGTAATCAGACGACACAGGAA	1689
	TTCCTGTGTCGTCTGATTACATCCTATTTCATCTTCAGCTGATG ACAAAGATGATAATGA**A**CTACATCTTGAAAAACATATTGGAGTA TCTTCTACACAATAAGTCTGTATTGTTTCTTGG	1690
	AGATGTAG**T**TCATTATC	1691
	GATAATGA**A**CTACATCT	1692
Predisposition to Colorectal Cancer Glu1317Gln GAA-CAA	GATTCTGCTAATACCCTGCAAATAGCAGAAATAAAAGAAAAGA TTGGAACTAGGTCAGCT**G**AAGATCCTGTGAGCGAAGTTCCAG CAGTGTCACAGCACCCTAGAACCAAATCCAGCAGAC	1693
	GTCTGCTGGATTTGGTTCTAGGGTGCTGTGACACTGCTGGAA CTTCGCTCACAGGATCTT**C**AGCTGACCTAGTTCCAATCTTTTC TTTTATTTCTGCTATTTGCAGGGTATTAGCAGAATC	1694
	GGTCAGCT**G**AAGATCCT	1695
	AGGATCTT**C**AGCTGACC	1696
Adenomatous polyposis coli Gln1328Term CAG-TAG	AAAGAAAAGATTGGAACTAGGTCAGCTGAAGATCCTGTGAGC GAAGTTCCAGCAGTGTCA**C**AGCACCCTAGAACCAAATCCAGC AGACTGCAGGGTTCTAGTTTATCTTCAGAATCAGCCA	1697
	TGGCTGATTCTGAAGATAAACTAGAACCCTGCAGTCTGCTGG ATTTGGTTCTAGGGTGCT**G**TGACACTGCTGGAACTTCGCTCA CAGGATCTTCAGCTGACCTAGTTCCAATCTTTTCTTT	1698
	CAGTGTCA**C**AGCACCCT	1699
	AGGGTGCT**G**TGACACTG	1700

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Gln1338Term CAG-TAG	GATCCTGTGAGCGAAGTTCCAGCAGTGTCACAGCACCCTAGA ACCAAATCCAGCAGACTGCAGGGTTCTAGTTTATCTTCAGAAT CAGCCAGGCACAAAGCTGTTGAATTTTCTTCAGGAG	1701
	CTCCTGAAGAAAATTCAACAGCTTTGTGCCTGGCTGATTCTGA AGATAAACTAGAACCCTGCAGTCTGCTGGATTTGGTTCTAGG GTGCTGTGACACTGCTGGAACTTCGCTCACAGGATC	1702
	GCAGACTGCAGGGTTCT	1703
	AGAACCCTGCAGTCTGC	1704
Adenomatous polyposis coli Leu1342Term TTA-TAA	AAGTTCCAGCAGTGTCACAGCACCCTAGAACCAAATCCAGCA GACTGCAGGGTTCTAGTTTATCTTCAGAATCAGCCAGGCACAA AGCTGTTGAATTTTCTTCAGGAGCGAAATCTCCCTC	1705
	GAGGGAGATTTCGCTCCTGAAGAAAATTCAACAGCTTTGTGC CTGGCTGATTCTGAAGATAAACTAGAACCCTGCAGTCTGCTG GATTTGGTTCTAGGGTGCTGTGACACTGCTGGAACTT	1706
	TTCTAGTTTATCTTCAG	1707
	CTGAAGATAAACTAGAA	1708
Adenomatous polyposis coli Arg1348Trp AGG-TGG	CAGCACCCTAGAACCAAATCCAGCAGACTGCAGGGTTCTAGT TTATCTTCAGAATCAGCCAGGCACAAAGCTGTTGAATTTTCTT CAGGAGCGAAATCTCCCTCCCGAAAGTGGTGCTCAG	1709
	CTGAGCACCACTTTCGGGAGGGAGATTTCGCTCCTGAAGAAA ATTCAACAGCTTTGTGCCTGGCTGATTCTGAAGATAAACTAGA ACCCTGCAGTCTGCTGGATTTGGTTCTAGGGTGCTG	1710
	AATCAGCCAGGCACAAA	1711
	TTTGTGCCTGGCTGATT	1712
Adenomatous polyposis coli Gly1357Term GGA-TGA	CTGCAGGGTTCTAGTTTATCTTCAGAATCAGCCAGGCACAAAG CTGTTGAATTTTCTTCAGGAGCGAAATCTCCCTCCCGAAAGTG GTGCTCAGACACCCCAAAGTCCACCTGAACACTAT	1713
	ATAGTGTTCAGGTGGACTTTGGGGTGTCTGAGCACCACTTTC GGGAGGGAGATTTCGCTCCTGAAGAAAATTCAACAGCTTTGT GCCTGGCTGATTCTGAAGATAAACTAGAACCCTGCAG	1714
	TTTCTTCAGGAGCGAAA	1715
	TTTCGCTCCTGAAGAAA	1716
Adenomatous polyposis coli Gln1367Term CAG-TAG	CCAGGCACAAAGCTGTTGAATTTTCTTCAGGAGCGAAATCTCC CTCCCGAAAGTGGTGCTCAGACACCCCAAAGTCCACCTGAAC ACTATGTTCAGGAGACCCCACTCATGTTTAGCAGAT	1717
	ATCTGCTAAACATGAGTGGGGTCTCCTGAACATAGTGTTCAG GTGGACTTTGGGGTGTCTGAGCACCACTTTCGGGAGGGAGAT TTCGCTCCTGAAGAAAATTCAACAGCTTTGTGCCTGG	1718
	GTGGTGCTCAGACACCC	1719
	GGGTGTCTGAGCACCAC	1720

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Lys1370Term AAA-TAA	AAAGCTGTTGAATTTTCTTCAGGAGCGAAATCTCCCTCCAAAA GTGGTGCTCAGACACCCAAAAGTCCACCTGAACACTATGTTC AGGAGACCCCACTCATGTTTAGCAGATGTACTTCTG	1721
	CAGAAGTACATCTGCTAAACATGAGTGGGGTCTCCTGAACATA GTGTTCAGGTGGACTTTTGGGTGTCTGAGCACCACTTTTGGA GGGAGATTTCGCTCCTGAAGAAAATTCAACAGCTTT	1722
	AGACACCCAAAAGTCCA	1723
	TGGACTTTTGGGTGTCT	1724
Adenomatous polyposis coli Ser1392Term TCA-TAA	CACCTGAACACTATGTTCAGGAGACCCCACTCATGTTTAGCA GATGTACTTCTGTCAGTTCACTTGATAGTTTTGAGAGTCGTTC GATTGCCAGCTCCGTTCAGAGTGAACCATGCAGTGG	1725
	CCACTGCATGGTTCACTCTGAACGGAGCTGGCAATCGAACGA CTCTCAAAACTATCAAGTGAACTGACAGAAGTACATCTGCTAA ACATGAGTGGGGTCTCCTGAACATAGTGTTCAGGTG	1726
	TGTCAGTTCACTTGATA	1727
	TATCAAGTGAACTGACA	1728
Adenomatous polyposis coli Ser1392Term TCA-TGA	CACCTGAACACTATGTTCAGGAGACCCCACTCATGTTTAGCA GATGTACTTCTGTCAGTTCACTTGATAGTTTTGAGAGTCGTTC GATTGCCAGCTCCGTTCAGAGTGAACCATGCAGTGG	1729
	CCACTGCATGGTTCACTCTGAACGGAGCTGGCAATCGAACGA CTCTCAAAACTATCAAGTGAACTGACAGAAGTACATCTGCTAA ACATGAGTGGGGTCTCCTGAACATAGTGTTCAGGTG	1730
	TGTCAGTTCACTTGATA	1731
	TATCAAGTGAACTGACA	1732
Adenomatous polyposis coli Glu1397Term GAG-TAG	GTTCAGGAGACCCCACTCATGTTTAGCAGATGTACTTCTGTCA GTTCACTTGATAGTTTTGAGAGTCGTTCGATTGCCAGCTCCGT TCAGAGTGAACCATGCAGTGGAATGGTAGGTGGCA	1733
	TGCCACCTACCATTCCACTGCATGGTTCACTCTGAACGGAGC TGGCAATCGAACGACTCTCAAAACTATCAAGTGAACTGACAGA AGTACATCTGCTAAACATGAGTGGGGTCTCCTGAAC	1734
	ATAGTTTTGAGAGTCGT	1735
	ACGACTCTCAAAACTAT	1736
Adenomatous polyposis coli Lys1449Term AAG-TAG	CAAACCATGCCACCAAGCAGAAGTAAAACACCTCCACCACCT CCTCAAACAGCTCAAACCAAAGCGAGAAGTACCTAAAAATAAAG CACCTACTGCTGAAAAGAGAGAGAGTGGACCTAAGC	1737
	GCTTAGGTCCACTCTCTCTCTTTTCAGCAGTAGGTGCTTTATT TTTAGGTACTTCTCGCTTGGTTTGAGCTGTTTGAGGAGGTGGT GGAGGTGTTTTACTTCTGCTTGGTGGCATGGTTTG	1738
	CTCAAACCAAGCGAGAA	1739
	TTCTCGCTTGGTTTGAG	1740
Adenomatous polyposis coli Arg1450Term CGA-TGA	ACCATGCCACCAAGCAGAAGTAAAACACCTCCACCACCTCCT CAAACAGCTCAAACCAAGCGAGAAGTACCTAAAAATAAAGCAC CTACTGCTGAAAAGAGAGAGAGTGGACCTAAGCAAG	1741

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTTGCTTAGGTCCACTCTCTCTCTTTTCAGCAGTAGGTGCTTT ATTTTTAGGTACTTCTC**G**CTTGGTTTGAGCTGTTTGAGGAGGT GGTGGAGGTGTTTTACTTCTGCTTGGTGGCATGGT	1742
	AAACCAAG**C**GAGAAGTA	1743
	TACTTCTC**G**CTTGGTTT	1744
Adenomatous polyposis coli Ser1503Term TCA-TAA	CAGATGCTGATACTTTATTACATTTTGCCACGGAAAGTACTCC AGATGGATTTTCTTGTT**C**ATCCAGCCTGAGTGCTCTGAGCCTC GATGAGCCATTTATACAGAAAGATGTGGAATTAAG	1745
	CTTAATTCCACATCTTTCTGTATAAATGGCTCATCGAGGCTCA GAGCACTCAGGCTGGAT**G**AACAAGAAAATCCATCTGGAGTAC TTTCCGTGGCAAAATGTAATAAAGTATCAGCATCTG	1746
	TTCTTGTT**C**ATCCAGCC	1747
	GGCTGGAT**G**AACAAGAA	1748
Adenomatous polyposis coli Gln1529Term CAG-TAG	CTGAGCCTCGATGAGCCATTTATACAGAAAGATGTGGAATTAA GAATAATGCCTCCAGTT**C**AGGAAAATGACAATGGGAATGAAAC AGAATCAGAGCAGCCTAAAGAATCAAATGAAAACC	1749
	GGTTTTCATTTGATTCTTTAGGCTGCTCTGATTCTGTTTCATTC CCATTGTCATTTTCCT**G**AACTGGAGGCATTATTCTTAATTCCAC ATCTTTCTGTATAAATGGCTCATCGAGGCTCAG	1750
	CTCCAGTT**C**AGGAAAAT	1751
	ATTTTCCT**G**AACTGGAG	1752
Adenomatous polyposis coli Ser1539Term TCA-TAA	ATGTGGAATTAAGAATAATGCCTCCAGTTCAGGAAAATGACAA TGGGAATGAAACAGAAT**C**AGAGCAGCCTAAAGAATCAAATGAA AACCAAGAGAAAGAGGCAGAAAAAACTATTGATTC	1753
	GAATCAATAGTTTTTTCTGCCTCTTTCTCTTGGTTTTCATTTGA TTCTTTAGGCTGCTCT**G**ATTCTGTTTCATTCCCATTGTCATTTT CCTGAACTGGAGGCATTATTCTTAATTCCACAT	1754
	AACAGAAT**C**AGAGCAGC	1755
	GCTGCTCT**G**ATTCTGTT	1756
Adenomatous polyposis coli Ser1567Term TCA-TGA	AAAACCAAGAGAAAGAGGCAGAAAAAACTATTGATTCTGAAAA GGACCTATTAGATGATT**C**AGATGATGATGATATTGAAATACTA GAAGAATGTATTATTTCTGCCATGCCAACAAAGTC	1757
	GACTTTGTTGGCATGGCAGAAATAATACATTCTTCTAGTATTTC AATATCATCATCATCT**G**AATCATCTAATAGGTCCTTTTCAGAAT CAATAGTTTTTTCTGCCTCTTTCTCTTGGTTTT	1758
	AGATGATT**C**AGATGATG	1759
	CATCATCT**G**AATCATCT	1760
Adenomatous polyposis coli Asp1822Val GAC-GTC	AGAGAGTTTTCTCAGACAACAAAGATTCAAAGAAACAGAATTT GAAAAATAATTCCAAGG**A**CTTCAATGATAAGCTCCCAAATAAT GAAGATAGAGTCAGAGGAAGTTTTGCTTTTGATTC	1761

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAATCAAAAGCAAAACTTCCTCTGACTCTATCTTCATTATTTGG GAGCTTATCATTGAAGTCCTTGGAATTATTTTTCAAATTCTGTT TCTTTGAATCTTTGTTGTCTGAGAAAACTCTCT	1762
	TTCCAAGGACTTCAATG	1763
	CATTGAAGTCCTTGGAA	1764
Adenomatous polyposis coli Leu2839Phe CTT-TTT	AAAACTGACAGCACAGAATCCAGTGGAACCCAAAGTCCTAAG CGCCATTCTGGGTCTTACCTTGTGACATCTGTTTAAAAGAGAG GAAGAATGAAACTAAGAAAATTCTATGTTAATTACA	1765
	TGTAATTAACATAGAATTTTCTTAGTTTCATTCTTCCTCTCTTTT AAACAGATGTCACAAGGTAAGACCCAGAATGGCGCTTAGGAC TTTGGGTTCCACTGGATTCTGTGCTGTCAGTTTT	1766
	GGTCTTACCTTGTGACA	1767
	TGTCACAAGGTAAGACC	1768

EXAMPLE 12
Parahemophilia - Factor V Deficiency

Deficiency in clotting Factor V is associated with a lifelong predisposition to thrombosis. The disease typically manifests itself with usually mild bleeding, although bleeding times and clotting times are consistently prolonged. Individuals that are heterozygous for a mutation in Factor V have lowered levels of factor V but probably never have abnormal bleeding. A large number of alleles with a range of presenting symptoms have been identified. The attached table discloses the correcting oligonucleotide base sequences for the Factor V oligonucleotides of the invention.

Table 19
Factor V Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Factor V deficiency Ala221Val GCC-GTC	TTGACTGAATGCTTATTTTGGCCTGTGTCTCTCCCTCTTTCTCA GATATAACAGTTTGTGCCCATGACCACATCAGCTGGCATCTGC TGGGAATGAGCTCGGGGCCAGAATTATTCTCCAT	1768
	ATGGAGAATAATTCTGGCCCCGAGCTCATTCCCAGCAGATGC CAGCTGATGTGGTCATGGGCACAAACTGTTATATCTGAGAAAG AGGGAGAGACACAGGCCAAAATAAGCATTCAGTCAA	1769
	AGTTTGTGCCCATGACC	1770
	GGTCATGGGCACAAACT	1771
Thrombosis Arg306Gly AGG-GGG	TGTCCTAACTCAGCTGGGATGCAGGCTTACATTGACATTAAAA ACTGCCCAAAGAAAACCAGGAATCTTAAGAAAATAACTCGTGA GCAGAGGCGGCACATGAAGAGGTGGGAATACTTCA	1772

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGAAGTATTCCCACCTCTTCATGTGCCGCCTCTGCTCACGAGT TATTTTCTTAAGATTCCTGGTTTTCTTTGGGCAGTTTTTAATGT CAATGTAAGCCTGCATCCCAGCTGAGTTAGGACA	1773
	AGAAAACCAAGGAATCTT	1774
	AAGATTCCTGGTTTTCT	1775
Thrombosis Arg306Thr AGG-ACG	GTCCTAACTCAGCTGGGATGCAGGCTTACATTGACATTAAAAA CTGCCCAAAGAAAACCAGGAATCTTAAGAAAATAACTCGTGAG CAGAGGCGGCACATGAAGAGGTGGGAATACTTCAT	1776
	ATGAAGTATTCCCACCTCTTCATGTGCCGCCTCTGCTCACGA GTTATTTTCTTAAGATTCCTGGTTTTCTTTGGGCAGTTTTTAAT GTCAATGTAAGCCTGCATCCCAGCTGAGTTAGGAC	1777
	GAAAACCAGGAATCTTA	1778
	TAAGATTCCTGGTTTTC	1779
Increased Risk Thrombosis Arg485Lys AGA-AAA	CCACAGAAAATGATGCCCAGTGCTTAACAAGACCATACTACAG TGACGTGGACATCATGAGAGACATCGCCTCTGGGCTAATAGG ACTACTTCTAATCTGTAAGAGCAGATCCCTGGACAG	1780
	CTGTCCAGGGATCTGCTCTTACAGATTAGAAGTAGTCCTATTA GCCCAGAGGCGATGTCTCTCATGATGTCCACGTCACTGTAGT ATGGTCTTGTTAAGCACTGGGCATCATTTTCTGTGG	1781
	CATCATGAGAGACATCG	1782
	CGATGTCTCTCATGATG	1783
Increased Risk Thrombosis Arg506Gln CGA-CAA	ACATCGCCTCTGGGCTAATAGGACTACTTCTAATCTGTAAGAG CAGATCCCTGGACAGGCGAGGAATACAGGTATTTTGTCCTTG AAGTAACCTTTCAGAAATTCTGAGAATTTCTTCTGG	1784
	CCAGAAGAAATTCTCAGAATTTCTGAAAGGTTACTTCAAGGAC AAAATACCTGTATTCCTCGCCTGTCCAGGGATCTGCTCTTACA GATTAGAAGTAGTCCTATTAGCCCAGAGGCGATGT	1785
	GGACAGGCGAGGAATAC	1786
	GTATTCCTCGCCTGTCC	1787
Factor V Deficiency Arg506Term CGA-TGA	GACATCGCCTCTGGGCTAATAGGACTACTTCTAATCTGTAAGA GCAGATCCCTGGACAGGCGAGGAATACAGGTATTTTGTCCTT GAAGTAACCTTTCAGAAATTCTGAGAATTTCTTCTG	1788
	CAGAAGAAATTCTCAGAATTTCTGAAAGGTTACTTCAAGGACA AAATACCTGTATTCCTCGCCTGTCCAGGGATCTGCTCTTACAG ATTAGAAGTAGTCCTATTAGCCCAGAGGCGATGTC	1789
	TGGACAGGCGAGGAATA	1790
	TATTCCTCGCCTGTCCA	1791
Thrombosis Arg712Term CGA-TGA	AGTGATGCTGACTATGATTACCAGAACAGACTGGCTGCAGCA TTAGGAATCAGGTCATTCCGAAACTCATCATTGAATCAGGAAG AAGAAGAGTTCAATCTTACTGCCCTAGCTCTGGAGA	1792
	TCTCCAGAGCTAGGGCAGTAAGATTGAACTCTTCTTCTTCCTG ATTCAATGATGAGTTTCGGAATGACCTGATTCCTAATGCTGCA GCCAGTCTGTTCTGGTAATCATAGTCAGCATCACT	1793
	GGTCATTCCGAAACTCA	1794

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGAGTTTCGGAATGACC	1795
Thrombosis His1299Arg CAT-CGT	TCAGTCAGACAAACCTTTCCCCAGCCCTCGGTCAGATGCCCA TTTCTCCAGACCTCAGCCATACAACCCTTTCTCTAGACTTCAG CCAGACAAACCTCTCTCCAGAACTCAGTCAAACAAA	1796
	TTTGTTTGACTGAGTTCTGGAGAGAGGTTTCTCTGGCTGAAGT CTAGAGAAAGGGTTGTATGGCTGAGGTCTGGAGAAATGGGCA TCTGACCGAGGGCTGGGGAAAGGTTTGTCTGACTGA	1797
	CCTCAGCCATACAACCC	1798
	GGGTTGTATGGCTGAGG	1799

EXAMPLE 13
Hemophilia - Factor VIII Deficiency

The attached table discloses the correcting oligonucleotide base sequences for the Factor VIII oligonucleotides of the invention.

Table 20
Factor VIII Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia A Tyr5Cys TAC-TGC	AGCTCTCCACCTGCTTCTTTCTGTGCCTTTTGCGATTCTGCTT TAGTGCCACCAGAAGATACTACCTGGGTGCAGTGGAACTGTC ATGGGACTATATGCAAAGTGATCTCGGTGAGCTGCC	1800
	GGCAGCTCACCGAGATCACTTTGCATATAGTCCCATGACAGT TCCACTGCACCCAGGTAGTATCTTCTGGTGGCACTAAAGCAG AATCGCAAAAGGCACAGAAAGAAGCAGGTGGAGAGCT	1801
	CAGAAGATACTACCTGG	1802
	CCAGGTAGTATCTTCTG	1803
Haemophilia A Leu7Arg CTG-CGG	CCACCTGCTTCTTTCTGTGCCTTTTGCGATTCTGCTTTAGTGC CACCAGAAGATACTACCTGGGTGCAGTGGAACTGTCATGGGA CTATATGCAAAGTGATCTCGGTGAGCTGCCTGTGGA	1804
	TCCACAGGCAGCTCACCGAGATCACTTTGCATATAGTCCCAT GACAGTTCCACTGCACCCAGGTAGTATCTTCTGGTGGCACTA AAGCAGAATCGCAAAAGGCACAGAAAGAAGCAGGTGG	1805
	ATACTACCTGGGTGCAG	1806
	CTGCACCCAGGTAGTAT	1807
Haemophilia A Ser(-1)Arg AGTg-AGG	AGTCATGCAAATAGAGCTCTCCACCTGCTTCTTTCTGTGCCTT TTGCGATTCTGCTTTAGTGCCACCAGAAGATACTACCTGGGT GCAGTGGAACTGTCATGGGACTATATGCAAAGTGAT	1808

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATCACTTTGCATATAGTCCCATGACAGTTCCACTGCACCCAG GTAGTATCTTCTGGTGGCACTAAAGCAGAATCGCAAAAGGCA CAGAAAGAAGCAGGTGGAGAGCTCTATTTGCATGACT	1809
	TGCTTTAGTGCCACCAG	1810
	CTGGTGGCACTAAAGCA	1811
Haemophilia A Arg(-5)Term gCGA-TGA	CATTTGTAGCAATAAGTCATGCAAATAGAGCTCTCCACCTGCT TCTTTCTGTGCCTTTTGCGATTCTGCTTTAGTGCCACCAGAAG ATACTACCTGGGTGCAGTGGAACTGTCATGGGACT	1812
	AGTCCCATGACAGTTCCACTGCACCCAGGTAGTATCTTCTGG TGGCACTAAAGCAGAATCGCAAAAGGCACAGAAAGAAGCAGG TGGAGAGCTCTATTTGCATGACTTATTGCTACAAATG	1813
	GCCTTTTGCGATTCTGC	1814
	GCAGAATCGCAAAAGGC	1815
Haemophilia A Glu11Val GAA-GTA	TTCTGTGCCTTTTGCGATTCTGCTTTAGTGCCACCAGAAGATA CTACCTGGGTGCAGTGGAACTGTCATGGGACTATATGCAAAG TGATCTCGGTGAGCTGCCTGTGGACGCAAGGTAAAG	1816
	CTTTACCTTGCGTCCACAGGCAGCTCACCGAGATCACTTTGC ATATAGTCCCATGACAGTTCCACTGCACCCAGGTAGTATCTTC TGGTGGCACTAAAGCAGAATCGCAAAAGGCACAGAA	1817
	TGCAGTGGAACTGTCAT	1818
	ATGACAGTTCCACTGCA	1819
Haemophilia A Trp14Gly aTGG-GGG	CTTTTGCGATTCTGCTTTAGTGCCACCAGAAGATACTACCTGG GTGCAGTGGAACTGTCATGGGACTATATGCAAAGTGATCTCG GTGAGCTGCCTGTGGACGCAAGGTAAAGGCATGTCC	1820
	GGACATGCCTTTACCTTGCGTCCACAGGCAGCTCACCGAGAT CACTTTGCATATAGTCCCATGACAGTTCCACTGCACCCAGGT AGTATCTTCTGGTGGCACTAAAGCAGAATCGCAAAAG	1821
	AACTGTCATGGGACTAT	1822
	ATAGTCCCATGACAGTT	1823
Haemophilia A Tyr46Term TACa-TAA	TTCACGCAGATTTCCTCCTAGAGTGCCAAAATCTTTTCCATTC AACACCTCAGTCGTGTACAAAAAGACTCTGTTTGTAGAATTCA CGGATCACCTTTTCAACATCGCTAAGCCAAGGCCA	1824
	TGGCCTTGGCTTAGCGATGTTGAAAAGGTGATCCGTGAATTC TACAAACAGAGTCTTTTTGTACACGACTGAGGTGTTGAATGGA AAAGATTTTGGCACTCTAGGAGGAAATCTGCGTGAA	1825
	GTCGTGTACAAAAAGAC	1826
	GTCTTTTTGTACACGAC	1827
Haemophilia A Asp56Glu GATc-GAA	ATCTTTTCCATTCAACACCTCAGTCGTGTACAAAAAGACTCTG TTTGTAGAATTCACGGATCACCTTTTCAACATCGCTAAGCCAA GGCCACCCTGGATGGGTAATGAAAACAATGTTGAA	1828

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTCAACATTGTTTTCATTACCCATCCAGGGTGGCCTTGGCTTA GCGATGTTGAAAAGGTGATCCGTGAATTCTACAAACAGAGTC TTTTTGTACACGACTGAGGTGTTGAATGGAAAAGAT	1829
	TTCACGGATCACCTTTT	1830
	AAAAGGTGATCCGTGAA	1831
Haemophilia A Gly73Val GGT-GTT	TTCTGGAGTACTATCCCCAAGTAACCTTTGGCGGACATCTCAT TCTTACAGGTCTGCTAGGTCCTACCATCCAGGCTGAGGTTTA TGATACAGTGGTCATTACACTTAAGAACATGGCTTC	1832
	GAAGCCATGTTCTTAAGTGTAATGACCACTGTATCATAAACCT CAGCCTGGATGGTAGGACCTAGCAGACCTGTAAGAATGAGAT GTCCGCCAAAGGTTACTTGGGGATAGTACTCCAGAA	1833
	TCTGCTAGGTCCTACCA	1834
	TGGTAGGACCTAGCAGA	1835
Haemophilia A Glu79Lys tGAG-AAG	CAAGTAACCTTTGGCGGACATCTCATTCTTACAGGTCTGCTAG GTCCTACCATCCAGGCTGAGGTTTATGATACAGTGGTCATTAC ACTTAAGAACATGGCTTCCCATCCTGTCAGTCTTC	1836
	GAAGACTGACAGGATGGGAAGCCATGTTCTTAAGTGTAATGA CCACTGTATCATAAACCTCAGCCTGGATGGTAGGACCTAGCA GACCTGTAAGAATGAGATGTCCGCCAAAGGTTACTTG	1837
	TCCAGGCTGAGGTTTAT	1838
	ATAAACCTCAGCCTGGA	1839
Haemophilia A Val80Asp GTT-GAT	TAACCTTTGGCGGACATCTCATTCTTACAGGTCTGCTAGGTCC TACCATCCAGGCTGAGGTTTATGATACAGTGGTCATTACACTT AAGAACATGGCTTCCCATCCTGTCAGTCTTCATGC	1840
	GCATGAAGACTGACAGGATGGGAAGCCATGTTCTTAAGTGTA ATGACCACTGTATCATAAACCTCAGCCTGGATGGTAGGACCT AGCAGACCTGTAAGAATGAGATGTCCGCCAAAGGTTA	1841
	GGCTGAGGTTTATGATA	1842
	TATCATAAACCTCAGCC	1843
Haemophilia A Asp82Val GAT-GTT	TTGGCGGACATCTCATTCTTACAGGTCTGCTAGGTCCTACCAT CCAGGCTGAGGTTTATGATACAGTGGTCATTACACTTAAGAAC ATGGCTTCCCATCCTGTCAGTCTTCATGCTGTTGG	1844
	CCAACAGCATGAAGACTGACAGGATGGGAAGCCATGTTCTTA AGTGTAATGACCACTGTATCATAAACCTCAGCCTGGATGGTA GGACCTAGCAGACCTGTAAGAATGAGATGTCCGCCAA	1845
	GGTTTATGATACAGTGG	1846
	CCACTGTATCATAAACC	1847
Haemophilia A Asp82Gly GAT-GGT	TTGGCGGACATCTCATTCTTACAGGTCTGCTAGGTCCTACCAT CCAGGCTGAGGTTTATGATACAGTGGTCATTACACTTAAGAAC ATGGCTTCCCATCCTGTCAGTCTTCATGCTGTTGG	1848

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCAACAGCATGAAGACTGACAGGATGGGAAGCCATGTTCTTA AGTGTAATGACCACTGTATCATAAACCTCAGCCTGGATGGTA GGACCTAGCAGACCTGTAAGAATGAGATGTCCGCCAA	1849
	GGTTTATGATACAGTGG	1850
	CCACTGTATCATAAACC	1851
Haemophilia A Val85Asp GTC-GAC	ATCTCATTCTTACAGGTCTGCTAGGTCCTACCATCCAGGCTGA GGTTTATGATACAGTGGTCATTACACTTAAGAACATGGCTTCC CATCCTGTCAGTCTTCATGCTGTTGGTGTATCCTA	1852
	TAGGATACACCAACAGCATGAAGACTGACAGGATGGGAAGCC ATGTTCTTAAGTGTAATGACCACTGTATCATAAACCTCAGCCT GGATGGTAGGACCTAGCAGACCTGTAAGAATGAGAT	1853
	TACAGTGGTCATTACAC	1854
	GTGTAATGACCACTGTA	1855
Haemophilia A Lys89Thr AAG-ACG	CAGGTCTGCTAGGTCCTACCATCCAGGCTGAGGTTTATGATA CAGTGGTCATTACACTTAAGAACATGGCTTCCCATCCTGTCA GTCTTCATGCTGTTGGTGTATCCTACTGGAAAGCTTC	1856
	GAAGCTTTCCAGTAGGATACACCAACAGCATGAAGACTGACA GGATGGGAAGCCATGTTCTTAAGTGTAATGACCACTGTATCAT AAACCTCAGCCTGGATGGTAGGACCTAGCAGACCTG	1857
	TACACTTAAGAACATGG	1858
	CCATGTTCTTAAGTGTA	1859
Haemophilia A Met91Val cATG-GTG	CTGCTAGGTCCTACCATCCAGGCTGAGGTTTATGATACAGTG GTCATTACACTTAAGAACATGGCTTCCCATCCTGTCAGTCTTC ATGCTGTTGGTGTATCCTACTGGAAAGCTTCTGAGG	1860
	CCTCAGAAGCTTTCCAGTAGGATACACCAACAGCATGAAGAC TGACAGGATGGGAAGCCATGTTCTTAAGTGTAATGACCACTG TATCATAAACCTCAGCCTGGATGGTAGGACCTAGCAG	1861
	TTAAGAACATGGCTTCC	1862
	GGAAGCCATGTTCTTAA	1863
Haemophilia A His94Arg CAT-CGT	CTACCATCCAGGCTGAGGTTTATGATACAGTGGTCATTACACT TAAGAACATGGCTTCCCATCCTGTCAGTCTTCATGCTGTTGGT GTATCCTACTGGAAAGCTTCTGAGGGTGAGTAAAA	1864
	TTTTACTCACCCTCAGAAGCTTTCCAGTAGGATACACCAACAG CATGAAGACTGACAGGATGGGAAGCCATGTTCTTAAGTGTAA TGACCACTGTATCATAAACCTCAGCCTGGATGGTAG	1865
	GGCTTCCCATCCTGTCA	1866
	TGACAGGATGGGAAGCC	1867
Haemophilia A His94Tyr cCAT-TAT	CCTACCATCCAGGCTGAGGTTTATGATACAGTGGTCATTACAC TTAAGAACATGGCTTCCCATCCTGTCAGTCTTCATGCTGTTGG TGTATCCTACTGGAAAGCTTCTGAGGGTGAGTAAA	1868

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTTACTCACCCTCAGAAGCTTTCCAGTAGGATACACCAACAGC ATGAAGACTGACAGGATGGGAAGCCATGTTCTTAAGTGTAAT GACCACTGTATCATAAACCTCAGCCTGGATGGTAGG	1869
	TGGCTTCCCATCCTGTC	1870
	GACAGGATGGGAAGCCA	1871
Haemophilia A Leu98Arg CTT-CGT	CTGAGGTTTATGATACAGTGGTCATTACACTTAAGAACATGGC TTCCCATCCTGTCAGTCTTCATGCTGTTGGTGTATCCTACTGG AAAGCTTCTGAGGGTGAGTAAAATACCCTCCTATT	1872
	AATAGGAGGGTATTTTACTCACCCTCAGAAGCTTTCCAGTAGG ATACACCAACAGCATGAAGACTGACAGGATGGGAAGCCATGT TCTTAAGTGTAATGACCACTGTATCATAAACCTCAG	1873
	TGTCAGTCTTCATGCTG	1874
	CAGCATGAAGACTGACA	1875
Haemophilia A Gly102Ser tGGT-AGT	GATACAGTGGTCATTACACTTAAGAACATGGCTTCCCATCCTG TCAGTCTTCATGCTGTTGGTGTATCCTACTGGAAAGCTTCTGA GGGTGAGTAAAATACCCTCCTATTGTCCTGTCATT	1876
	AATGACAGGACAATAGGAGGGTATTTTACTCACCCTCAGAAG CTTTCCAGTAGGATACACCAACAGCATGAAGACTGACAGGAT GGGAAGCCATGTTCTTAAGTGTAATGACCACTGTATC	1877
	ATGCTGTTGGTGTATCC	1878
	GGATACACCAACAGCAT	1879
Haemophilia A Glu113Asp GAAt-GAC	CTTTGAGTGTACAGTGGATATAGAAAGGACAATTTTATTTCTTC CTGCTATAGGAGCTGAATATGATGATCAGACCAGTCAAAGGG AGAAAGAAGATGATAAAGTCTTCCCTGGTGGAAGC	1880
	GCTTCCACCAGGGAAGACTTTATCATCTTCTTTCTCCCTTTGA CTGGTCTGATCATCATATTCAGCTCCTATAGCAGGAAGAAATA AAATTGTCCTTTCTATATCCACTGTACACTCAAAG	1881
	GGAGCTGAATATGATGA	1882
	TCATCATATTCAGCTCC	1883
Haemophilia A Tyr114Cys TAT-TGT	TTGAGTGTACAGTGGATATAGAAAGGACAATTTTATTTCTTCCT GCTATAGGAGCTGAATATGATGATCAGACCAGTCAAAGGGAG AAAGAAGATGATAAAGTCTTCCCTGGTGGAAGCCA	1884
	TGGCTTCCACCAGGGAAGACTTTATCATCTTCTTTCTCCCTTT GACTGGTCTGATCATCATATTCAGCTCCTATAGCAGGAAGAAA TAAAATTGTCCTTTCTATATCCACTGTACACTCAA	1885
	AGCTGAATATGATGATC	1886
	GATCATCATATTCAGCT	1887
Haemophilia A Asp116Gly GAT-GGT	GTACAGTGGATATAGAAAGGACAATTTTATTTCTTCCTGCTATA GGAGCTGAATATGATGATCAGACCAGTCAAAGGGAGAAAGAA GATGATAAAGTCTTCCCTGGTGGAAGCCATACATA	1888

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TATGTATGGCTTCCACCAGGGAAGACTTTATCATCTTCTTTCT CCCTTTGACTGGTCTGATCATCATATTCAGCTCCTATAGCAGG AAGAAATAAAATTGTCCTTTCTATATCCACTGTAC	1889
	ATATGATGATCAGACCA	1890
	TGGTCTGATCATCATAT	1891
Haemophilia A Gln117Term tCAG-TAG	ACAGTGGATATAGAAAGGACAATTTTATTTCTTCCTGCTATAG GAGCTGAATATGATGATCAGACCAGTCAAAGGGAGAAAGAAG ATGATAAAGTCTTCCCTGGTGGAAGCCATACATATG	1892
	CATATGTATGGCTTCCACCAGGGAAGACTTTATCATCTTCTTT CTCCCTTTGACTGGTCTGATCATCATATTCAGCTCCTATAGCA GGAAGAAATAAAATTGTCCTTTCTATATCCACTGT	1893
	ATGATGATCAGACCAGT	1894
	ACTGGTCTGATCATCAT	1895
Haemophilia A Thr118Ile ACC-ATC	TGGATATAGAAAGGACAATTTTATTTCTTCCTGCTATAGGAGC TGAATATGATGATCAGACCAGTCAAAGGGAGAAAGAAGATGA TAAAGTCTTCCCTGGTGGAAGCCATACATATGTCTG	1896
	CAGACATATGTATGGCTTCCACCAGGGAAGACTTTATCATCTT CTTTCTCCCTTTGACTGGTCTGATCATCATATTCAGCTCCTAT AGCAGGAAGAAATAAAATTGTCCTTTCTATATCCA	1897
	TGATCAGACCAGTCAAA	1898
	TTTGACTGGTCTGATCA	1899
Haemophilia A Glu122Term gGAG-TAG	AGGACAATTTTATTTCTTCCTGCTATAGGAGCTGAATATGATG ATCAGACCAGTCAAAGGGAGAAAGAAGATGATAAAGTCTTCC CTGGTGGAAGCCATACATATGTCTGGCAGGTCCTGA	1900
	TCAGGACCTGCCAGACATATGTATGGCTTCCACCAGGGAAGA CTTTATCATCTTCTTTCTCCCTTTGACTGGTCTGATCATCATAT TCAGCTCCTATAGCAGGAAGAAATAAAATTGTCCT	1901
	GTCAAAGGGAGAAAGAA	1902
	TTCTTTCTCCCTTTGAC	1903
Haemophilia A Asp126His tGAT-CAT	TTTCTTCCTGCTATAGGAGCTGAATATGATGATCAGACCAGTC AAAGGGAGAAAGAAGATGATAAAGTCTTCCCTGGTGGAAGCC ATACATATGTCTGGCAGGTCCTGAAAGAGAATGGTC	1904
	GACCATTCTCTTTCAGGACCTGCCAGACATATGTATGGCTTCC ACCAGGGAAGACTTTATCATCTTCTTTCTCCCTTTGACTGGTC TGATCATCATATTCAGCTCCTATAGCAGGAAGAAA	1905
	AAGAAGATGATAAAGTC	1906
	GACTTTATCATCTTCTT	1907
Haemophilia A Gln139Term gCAG-TAG	AGTCAAAGGGAGAAAGAAGATGATAAAGTCTTCCCTGGTGGA AGCCATACATATGTCTGGCAGGTCCTGAAAGAGAATGGTCCA ATGGCCTCTGACCCACTGTGCCTTACCTACTCATATC	1908

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GATATGAGTAGGTAAGGCACAGTGGGTCAGAGGCCATTGGA CCATTCTCTTTCAGGACCTGCCAGACATATGTATGGCTTCCAC CAGGGAAGACTTTATCATCTTCTTTCTCCCTTTGACT	1909
	ATGTCTGGCAGGTCCTG	1910
	CAGGACCTGCCAGACAT	1911
Haemophilia A Val140Ala GTC-GCC	AAAGGGAGAAAGAAGATGATAAAGTCTTCCCTGGTGGAAGCC ATACATATGTCTGGCAGGTCCTGAAAGAGAATGGTCCAATGG CCTCTGACCCACTGTGCCTTACCTACTCATATCTTTC	1912
	GAAAGATATGAGTAGGTAAGGCACAGTGGGTCAGAGGCCATT GGACCATTCTCTTTCAGGACCTGCCAGACATATGTATGGCTT CCACCAGGGAAGACTTTATCATCTTCTTTCTCCCTTT	1913
	CTGGCAGGTCCTGAAAG	1914
	CTTTCAGGACCTGCCAG	1915
Haemophilia A Asn144Lys AATg-AAA	AGATGATAAAGTCTTCCCTGGTGGAAGCCATACATATGTCTG GCAGGTCCTGAAAGAGAATGGTCCAATGGCCTCTGACCCACT GTGCCTTACCTACTCATATCTTTCTCATGTGGACCTG	1916
	CAGGTCCACATGAGAAAGATATGAGTAGGTAAGGCACAGTGG GTCAGAGGCCATTGGACCATTCTCTTTCAGGACCTGCCAGAC ATATGTATGGCTTCCACCAGGGAAGACTTTATCATCT	1917
	AAAGAGAATGGTCCAAT	1918
	ATTGGACCATTCTCTTT	1919
Haemophilia AG Gly145Asp GGT-GAT	ATGATAAAGTCTTCCCTGGTGGAAGCCATACATATGTCTGGCA GGTCCTGAAAGAGAATGGGTCCAATGGCCTCTGACCCACTGTG CCTTACCTACTCATATCTTTCTCATGTGGACCTGGT	1920
	ACCAGGTCCACATGAGAAAGATATGAGTAGGTAAGGCACAGT GGGTCAGAGGCCATTGGACCATTCTCTTTCAGGACCTGCCAG ACATATGTATGGCTTCCACCAGGGAAGACTTTATCAT	1921
	AGAGAATGGGTCCAATGG	1922
	CCATTGGACCATTCTCT	1923
Haemophilia A Gly145Val GGT-GTT	ATGATAAAGTCTTCCCTGGTGGAAGCCATACATATGTCTGGCA GGTCCTGAAAGAGAATGGGTCCAATGGCCTCTGACCCACTGTG CCTTACCTACTCATATCTTTCTCATGTGGACCTGGT	1924
	ACCAGGTCCACATGAGAAAGATATGAGTAGGTAAGGCACAGT GGGTCAGAGGCCATTGGACCATTCTCTTTCAGGACCTGCCAG ACATATGTATGGCTTCCACCAGGGAAGACTTTATCAT	1925
	AGAGAATGGGTCCAATGG	1926
	CCATTGGACCATTCTCT	1927
Haemophilia A Pro146Ser tCCA-TCA	GATAAAGTCTTCCCTGGTGGAAGCCATACATATGTCTGGCAG GTCCTGAAAGAGAATGGTCCAATGGCCTCTGACCCACTGTGC CTTACCTACTCATATCTTTCTCATGTGGACCTGGTAA	1928

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTACCAGGTCCACATGAGAAAGATATGAGTAGGTAAGGCACA GTGGGTCAGAGGCCATTGGACCATTCTCTTTCAGGACCTGCC AGACATATGTATGGCTTCCACCAGGGAAGACTTTATC	1929
	AGAATGGTCCAATGGCC	1930
	GGCCATTGGACCATTCT	1931
Haemophilia A Cys153Trp TGCc-TGG	CCATACATATGTCTGGCAGGTCCTGAAAGAGAATGGTCCAAT GGCCTCTGACCCACTGTGCCTTACCTACTCATATCTTTCTCAT GTGGACCTGGTAAAAGACTTGAATTCAGGCCTCATT	1932
	AATGAGGCCTGAATTCAAGTCTTTTACCAGGTCCACATGAGAA AGATATGAGTAGGTAAGGCACAGTGGGTCAGAGGCCATTGGA CCATTCTCTTTCAGGACCTGCCAGACATATGTATGG	1933
	CCACTGTGCCTTACCTA	1934
	TAGGTAAGGCACAGTGG	1935
Haemophilia A Tyr156Term TACt-TAA	TGTCTGGCAGGTCCTGAAAGAGAATGGTCCAATGGCCTCTGA CCCACTGTGCCTTACCTACTCATATCTTTCTCATGTGGACCTG GTAAAAGACTTGAATTCAGGCCTCATTGGAGCCCTA	1936
	TAGGGCTCCAATGAGGCCTGAATTCAAGTCTTTTACCAGGTC CACATGAGAAAGATATGAGTAGGTAAGGCACAGTGGGTCAGA GGCCATTGGACCATTCTCTTTCAGGACCTGCCAGACA	1937
	CTTACCTACTCATATCT	1938
	AGATATGAGTAGGTAAG	1939
Haemophilia A Ser157Pro cTCA-CCA	GTCTGGCAGGTCCTGAAAGAGAATGGTCCAATGGCCTCTGAC CCACTGTGCCTTACCTACTCATATCTTTCTCATGTGGACCTGG TAAAAGACTTGAATTCAGGCCTCATTGGAGCCCTAC	1940
	GTAGGGCTCCAATGAGGCCTGAATTCAAGTCTTTTACCAGGT CCACATGAGAAAGATATGAAGTAGGTAAGGCACAGTGGGTCAG AGGCCATTGGACCATTCTCTTTCAGGACCTGCCAGAC	1941
	TTACCTACTCATATCTT	1942
	AAGATATGAAGTAGGTAA	1943
Haemophilia A Ser160Pro tTCT-CCT	GTCCTGAAAGAGAATGGTCCAATGGCCTCTGACCCACTGTGC CTTACCTACTCATATCTTTCTCATGTGGACCTGGTAAAAGACT TGAATTCAGGCCTCATTGGAGCCCTACTAGTATGTA	1944
	TACATACTAGTAGGGCTCCAATGAGGCCTGAATTCAAGTCTTT TACCAGGTCCACATGAGAAAGATATGAGTAGGTAAGGCACAG TGGGTCAGAGGCCATTGGACCATTCTCTTTCAGGAC	1945
	CATATCTTTCTCATGTG	1946
	CACATGAGAAAGATATG	1947
Haemophilia A Val162Met tGTG-ATG	AAAGAGAATGGTCCAATGGCCTCTGACCCACTGTGCCTTACC TACTCATATCTTTCTCATGTGGACCTGGTAAAAGACTTGAATT CAGGCCTCATTGGAGCCCTACTAGTATGTAGAGAAG	1948

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTTCTCTACATACTAGTAGGGCTCCAATGAGGCCTGAATTCAA GTCTTTTACCAGGTCCACATGAGAAAGATATGAGTAGGTAAG GCACAGTGGGTCAGAGGCCATTGGACCATTCTCTTT	1949
	TTTCTCATGTGGACCTG	1950
	CAGGTCCACATGAGAAA	1951
Haemophilia A Lys166Thr AAA-ACA	CAATGGCCTCTGACCCACTGTGCCTTACCTACTCATATCTTTC TCATGTGGACCTGGTAAAAGACTTGAATTCAGGCCTCATTGG AGCCCTACTAGTATGTAGAGAAGGTAAGTGTATGAA	1952
	TTCATACACTTACCTTCTCTACATACTAGTAGGGCTCCAATGA GGCCTGAATTCAAGTCTTTTACCAGGTCCACATGAGAAAGATA TGAGTAGGTAAGGCACAGTGGGTCAGAGGCCATTG	1953
	CCTGGTAAAAGACTTGA	1954
	TCAAGTCTTTTACCAGG	1955
Haemophilia A Ser170Leu TCA-TTA	ACCCACTGTGCCTTACCTACTCATATCTTTCTCATGTGGACCT GGTAAAAGACTTGAATTCAGGCCTCATTGGAGCCCTACTAGT ATGTAGAGAAGGTAAGTGTATGAAAGCGTAGGATTG	1956
	CAATCCTACGCTTTCATACACTTACCTTCTCTACATACTAGTAG GGCTCCAATGAGGCCTGAATTCAAGTCTTTTACCAGGTCCAC ATGAGAAAGATATGAGTAGGTAAGGCACAGTGGGT	1957
	CTTGAATTCAGGCCTCA	1958
	TGAGGCCTGAATTCAAG	1959
Haemophilia A Phe195Val aTTT-GTT	AATGTTCTCACTTCTTTTTCAGGGAGTCTGGCCAAGGAAAAGA CACAGACCTTGCACAAATTTATACTACTTTTTGCTGTATTTGAT GAAGGTTAGTGAGTCTTAATCTGAATTTTGGATT	1960
	AATCCAAAATTCAGATTAAGACTCACTAACCTTCATCAAATACA GCAAAAAGTAGTATAAATTTGTGCAAGGTCTGTGTCTTTTCCT TGGCCAGACTCCCTGAAAAAGAAGTGAGAACATT	1961
	TGCACAAATTTATACTA	1962
	TAGTATAAATTTGTGCA	1963
Haemophilia A Leu198His CTT-CAT	CTTCTTTTTCAGGGAGTCTGGCCAAGGAAAAGACACAGACCT TGCACAAATTTATACTACTTTTTGCTGTATTTGATGAAGGTTAG TGAGTCTTAATCTGAATTTTGGATTCCTGAAAGAA	1964
	TTCTTTCAGGAATCCAAAATTCAGATTAAGACTCACTAACCTTC ATCAAATACAGCAAAAAGTAGTATAAATTTGTGCAAGGTCTGT GTCTTTTCCTTGGCCAGACTCCCTGAAAAAGAAG	1965
	TATACTACTTTTTGCTG	1966
	CAGCAAAAAGTAGTATA	1967
Haemophilia A Ala200Asp GCT-GAT	TTTCAGGGAGTCTGGCCAAGGAAAAGACACAGACCTTGCACA AATTTATACTACTTTTTGCTGTATTTGATGAAGGTTAGTGAGTC TTAATCTGAATTTTGGATTCCTGAAAGAAATCCTC	1968

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAGGATTTCTTTCAGGAATCCAAAATTCAGATTAAGACTCACT AACCTTCATCAAATACAGCAAAAAGTAGTATAAATTTGTGCAA GGTCTGTGTCTTTTCCTTGGCCAGACTCCCTGAAA	1969
	ACTTTTTGCTGTATTTG	1970
	CAAATACAGCAAAAAGT	1971
Haemophilia A Ala200Thr tGCT-ACT	TTTTCAGGGAGTCTGGCCAAGGAAAAGACACAGACCTTGCAC AAATTTATACTACTTTTTGCTGTATTTGATGAAGGTTAGTGAGT CTTAATCTGAATTTTGGATTCCTGAAAGAAATCCT	1972
	AGGATTTCTTTCAGGAATCCAAAATTCAGATTAAGACTCACTA ACCTTCATCAAATACAGCAAAAAGTAGTATAAATTTGTGCAAG GTCTGTGTCTTTTCCTTGGCCAGACTCCCTGAAAA	1973
	TACTTTTTGCTGTATTT	1974
	AAATACAGCAAAAAGTA	1975
Haemophilia A Val234Phe aGTC-TTC	AACTCCTTGATGCAGGATAGGGATGCTGCATCTGCTCGGGCC TGGCCTAAAATGCACACAGTCAATGGTTATGTAAACAGGTCTC TGCCAGGTATGTACACACCTGCTCAACAATCCTCAG	1976
	CTGAGGATTGTTGAGCAGGTGTGTACATACCTGGCAGAGACC TGTTTACATAACCATTGACTGTGTGCATTTTAGGCCAGGCCCG AGCAGATGCAGCATCCCTATCCTGCATCAAGGAGTT	1977
	TGCACACAGTCAATGGT	1978
	ACCATTGACTGTGTGCA	1979
Haemophilia A Gly247Glu GGA-GAA	ATTTCAGATTCTCTACTTCATAGCCATAGGTGTCTTATTCCTAC TTTACAGGTCTGATTGGATGCCACAGGAAATCAGTCTATTGGC ATGTGATTGGAATGGGCACCACTCCTGAAGTGCA	1980
	TGCACTTCAGGAGTGGTGCCCATTCCAATCACATGCCAATAG ACTGATTTCCTGTGGCATCCAATCAGACCTGTAAAGTAGGAAT AAGACACCTATGGCTATGAAGTAGAGAATCTGAAAT	1981
	TCTGATTGGATGCCACA	1982
	TGTGGCATCCAATCAGA	1983
Haemophilia A Trp255Cys TGGc-TGT	ATAGGTGTCTTATTCCTACTTTACAGGTCTGATTGGATGCCAC AGGAAATCAGTCTATTGGCATGTGATTGGAATGGGCACCACT CCTGAAGTGCACTCAATATTCCTCGAAGGTCACACA	1984
	TGTGTGACCTTCGAGGAATATTGAGTGCACTTCAGGAGTGGT GCCCATTCCAATCACATGCCAATAGACTGATTTCCTGTGGCAT CCAATCAGACCTGTAAAGTAGGAATAAGACACCTAT	1985
	GTCTATTGGCATGTGAT	1986
	ATCACATGCCAATAGAC	1987
Haemophilia A Trp255Term TGGc-TGA	ATAGGTGTCTTATTCCTACTTTACAGGTCTGATTGGATGCCAC AGGAAATCAGTCTATTGGCATGTGATTGGAATGGGCACCACT CCTGAAGTGCACTCAATATTCCTCGAAGGTCACACA	1988

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGTGTGACCTTCGAGGAATATTGAGTGCACTTCAGGAGTGGT GCCCATTCCAATCACATGCCAATAGACTGATTTCCTGTGGCAT CCAATCAGACCTGTAAAGTAGGAATAAGACACCTAT	1989
	GTCTATTGGCATGTGAT	1990
	ATCACATGCCAATAGAC	1991
Haemophilia A His256Leu CAT-CTT	AGGTGTCTTATTCCTACTTTACAGGTCTGATTGGATGCCACAG GAAATCAGTCTATTGGCATGTGATTGGAATGGGCACCACTCC TGAAGTGCACTCAATATTCCTCGAAGGTCACACATT	1992
	AATGTGTGACCTTCGAGGAATATTGAGTGCACTTCAGGAGTG GTGCCCATTCCAATCACATGCCAATAGACTGATTTCCTGTGG CATCCAATCAGACCTGTAAAGTAGGAATAAGACACCT	1993
	CTATTGGCATGTGATTG	1994
	CAATCACATGCCAATAG	1995
Haemophilia A Gly259Arg tGGA-AGA	TATTCCTACTTTACAGGTCTGATTGGATGCCACAGGAAATCAG TCTATTGGCATGTGATTGGAATGGGCACCACTCCTGAAGTGC ACTCAATATTCCTCGAAGGTCACACATTTCTTGTGA	1996
	TCACAAGAAATGTGTGACCTTCGAGGAATATTGAGTGCACTTC AGGAGTGGTGCCCATTCCAATCACATGCCAATAGACTGATTT CCTGTGGCATCCAATCAGACCTGTAAAGTAGGAATA	1997
	ATGTGATTGGAATGGGC	1998
	GCCCATTCCAATCACAT	1999
Haemophilia A Val266Gly GTG-GGG	TTGGATGCCACAGGAAATCAGTCTATTGGCATGTGATTGGAAT GGGCACCACTCCTGAAGTGCACTCAATATTCCTCGAAGGTCA CACATTTCTTGTGAGGAACCATCGCCAGGCGTCCTT	2000
	AAGGACGCCTGGCGATGGTTCCTCACAAGAAATGTGTGACCT TCGAGGAATATTGAGTGCACTTCAGGAGTGGTGCCCATTCCA ATCACATGCCAATAGACTGATTTCCTGTGGCATCCAA	2001
	TCCTGAAGTGCACTCAA	2002
	TTGAGTGCACTTCAGGA	2003
Haemophilia A Glu272Gly GAA-GGA	CAGTCTATTGGCATGTGATTGGAATGGGCACCACTCCTGAAG TGCACTCAATATTCCTCGAAGGTCACACATTTCTTGTGAGGAA CCATCGCCAGGCGTCCTTGGAAATCTCGCCAATAAC	2004
	GTTATTGGCGAGATTTCCAAGGACGCCTGGCGATGGTTCCTC ACAAGAAATGTGTGACCTTCGAGGAATATTGAGTGCACTTCAG GAGTGGTGCCCATTCCAATCACATGCCAATAGACTG	2005
	ATTCCTCGAAGGTCACA	2006
	TGTGACCTTCGAGGAAT	2007
Haemophilia A Glu272Lys cGAA-AAA	TCAGTCTATTGGCATGTGATTGGAATGGGCACCACTCCTGAA GTGCACTCAATATTCCTCGAAGGTCACACATTTCTTGTGAGGA ACCATCGCCAGGCGTCCTTGGAAATCTCGCCAATAA	2008

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTATTGGCGAGATTTCCAAGGACGCCTGGCGATGGTTCCTCA CAAGAAATGTGTGACCTTCGAGGAATATTGAGTGCACTTCAG GAGTGGTGCCCATTCCAATCACATGCCAATAGACTGA	2009
	TATTCCTCGAAGGTCAC	2010
	GTGACCTTCGAGGAATA	2011
Haemophilia A Thr275Ile ACA-ATA	GGCATGTGATTGGAATGGGCACCACTCCTGAAGTGCACTCAA TATTCCTCGAAGGTCACACATTTCTTGTGAGGAACCATCGCCA GGCGTCCTTGGAAATCTCGCCAATAACTTTCCTTAC	2012
	GTAAGGAAAGTTATTGGCGAGATTTCCAAGGACGCCTGGCGA TGGTTCCTCACAAGAAATGTGTGACCTTCGAGGAATATTGAGT GCACTTCAGGAGTGGTGCCCATTCCAATCACATGCC	2013
	AGGTCACACATTTCTTG	2014
	CAAGAAATGTGTGACCT	2015
Haemophilia A Val278Ala GTG-GCG	TTGGAATGGGCACCACTCCTGAAGTGCACTCAATATTCCTCG AAGGTCACACATTTCTTGTGAGGAACCATCGCCAGGCGTCCT TGGAAATCTCGCCAATAACTTTCCTTACTGCTCAAAC	2016
	GTTTGAGCAGTAAGGAAAGTTATTGGCGAGATTTCCAAGGAC GCCTGGCGATGGTTCCTCACAAGAAATGTGTGACCTTCGAGG AATATTGAGTGCACTTCAGGAGTGGTGCCCATTCCAA	2017
	ATTTCTTGTGAGGAACC	2018
	GGTTCCTCACAAGAAAT	2019
Haemophilia A Asn280Ile AAC-ATC	TGGGCACCACTCCTGAAGTGCACTCAATATTCCTCGAAGGTC ACACATTTCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAA TCTCGCCAATAACTTTCCTTACTGCTCAAACACTCTT	2020
	AAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGATTTCCA AGGACGCCTGGCGATGGTTCCTCACAAGAAATGTGTGACCTT CGAGGAATATTGAGTGCACTTCAGGAGTGGTGCCCA	2021
	TGTGAGGAACCATCGCC	2022
	GGCGATGGTTCCTCACA	2023
Haemophilia A Arg282Cys tCGC-TGC	ACCACTCCTGAAGTGCACTCAATATTCCTCGAAGGTCACACAT TTCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAATCTCGC CAATAACTTTCCTTACTGCTCAAACACTCTTGATGG	2024
	CCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGA TTTCCAAGGACGCCTGGCGATGGTTCCTCACAAGAAATGTGT GACCTTCGAGGAATATTGAGTGCACTTCAGGAGTGGT	2025
	GGAACCATCGCCAGGCG	2026
	CGCCTGGCGATGGTTCC	2027
Haemophilia A Arg282His CGC-CAC	CCACTCCTGAAGTGCACTCAATATTCCTCGAAGGTCACACATT TCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAATCTCGCC AATAACTTTCCTTACTGCTCAAACACTCTTGATGGA	2028

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAG ATTTCCAAGGACGCCTGG**C**GATGGTTCCTCACAAGAAATGTG TGACCTTCGAGGAATATTGAGTGCACTTCAGGAGTGG	2029
	GAACCATC**G**CCAGGCGT	2030
	ACGCCTGG**C**GATGGTTC	2031
Haemophilia A Arg282Leu CGC-CTC	CCACTCCTGAAGTGCACTCAATATTCCTCGAAGGTCACACATT TCTTGTGAGGAACCATC**G**CCAGGCGTCCTTGGAAATCTCGCC AATAACTTTCCTTACTGCTCAAACACTCTTGATGGA	2032
	TCCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAG ATTTCCAAGGACGCCTGG**C**GATGGTTCCTCACAAGAAATGTG TGACCTTCGAGGAATATTGAGTGCACTTCAGGAGTGG	2033
	GAACCATC**G**CCAGGCGT	2034
	ACGCCTGG**C**GATGGTTC	2035
Haemophilia A Ala284Glu GCG-GAG	CTGAAGTGCACTCAATATTCCTCGAAGGTCACACATTTCTTGT GAGGAACCATCGCCAGG**C**GTCCTTGGAAATCTCGCCAATAAC TTTCCTTACTGCTCAAACACTCTTGATGGACCTTGG	2036
	CCAAGGTCCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATT GGCGAGATTTCCAAGGAC**G**CCTGGCGATGGTTCCTCACAAG AAATGTGTGACCTTCGAGGAATATTGAGTGCACTTCAG	2037
	TCGCCAGG**C**GTCCTTGG	2038
	CCAAGGAC**G**CCTGGCGA	2039
Haemophilia A Ala284Pro gGCG-CCG	CCTGAAGTGCACTCAATATTCCTCGAAGGTCACACATTTCTTG TGAGGAACCATCGCCAG**G**CGTCCTTGGAAATCTCGCCAATAA CTTTCCTTACTGCTCAAACACTCTTGATGGACCTTG	2040
	CAAGGTCCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTG GCGAGATTTCCAAGGACG**C**CTGGCGATGGTTCCTCACAAGAA ATGTGTGACCTTCGAGGAATATTGAGTGCACTTCAGG	2041
	ATCGCCAG**G**CGTCCTTG	2042
	CAAGGACG**C**CTGGCGAT	2043
Haemophilia A Ser289Leu TCG-TTG	TATTCCTCGAAGGTCACACATTTCTTGTGAGGAACCATCGCCA GGCGTCCTTGGAAATCT**C**GCCAATAACTTTCCTTACTGCTCAA ACACTCTTGATGGACCTTGGACAGTTTCTACTGTT	2044
	AACAGTAGAAACTGTCCAAGGTCCATCAAGAGTGTTTGAGCA GTAAGGAAAGTTATTGGC**G**AGATTTCCAAGGACGCCTGGCGA TGGTTCCTCACAAGAAATGTGTGACCTTCGAGGAATA	2045
	GGAAATCT**C**GCCAATAA	2046
	TTATTGGC**G**AGATTTCC	2047
Haemophilia A Phe293Ser TTC-TCC	GTCACACATTTCTTGTGAGGAACCATCGCCAGGCGTCCTTGG AAATCTCGCCAATAACTTT**T**CCTTACTGCTCAAACACTCTTGAT GGACCTTGGACAGTTTCTACTGTTTTGTCATATCTC	2048

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAGATATGACAAAACAGTAGAAACTGTCCAAGGTCCATCAAG AGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGATTTCCAAG GACGCCTGGCGATGGTTCCTCACAAGAAATGTGTGAC	2049
	AATAACTTTCCTTACTG	2050
	CAGTAAGGAAAGTTATT	2051
Haemophilia A Thr295Ala tACT-GCT	ACATTTCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAATC TCGCCAATAACTTTCCTTACTGCTCAAACACTCTTGATGGACC TTGGACAGTTTCTACTGTTTTGTCATATCTCTTCCC	2052
	GGGAAGAGATATGACAAAACAGTAGAAACTGTCCAAGGTCCA TCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGATTTC CAAGGACGCCTGGCGATGGTTCCTCACAAGAAATGT	2053
	CTTTCCTTACTGCTCAA	2054
	TTGAGCAGTAAGGAAAG	2055
Haemophilia A Thr295Ile ACT-ATT	CATTTCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAATCT CGCCAATAACTTTCCTTACTGCTCAAACACTCTTGATGGACCT TGGACAGTTTCTACTGTTTTGTCATATCTCTTCCCA	2056
	TGGGAAGAGATATGACAAAACAGTAGAAACTGTCCAAGGTCC ATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGATTT CCAAGGACGCCTGGCGATGGTTCCTCACAAGAAATG	2057
	TTTCCTTACTGCTCAAA	2058
	TTTGAGCAGTAAGGAAA	2059
Haemophilia A Ala296Val GCT-GTT	TTCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAATCTCGC CAATAACTTTCCTTACTGCTCAAACACTCTTGATGGACCTTGG ACAGTTTCTACTGTTTTGTCATATCTCTTCCCACCA	2060
	TGGTGGGAAGAGATATGACAAAACAGTAGAAACTGTCCAAGG TCCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAG ATTTCCAAGGACGCCTGGCGATGGTTCCTCACAAGAA	2061
	CCTTACTGCTCAAACAC	2062
	GTGTTTGAGCAGTAAGG	2063
Haemophilia A Leu308Pro CTG-CCG	TCTCGCCAATAACTTTCCTTACTGCTCAAACACTCTTGATGGA CCTTGGACAGTTTCTACTGTTTTGTCATATCTCTTCCCACCAA CATGGTAATATCTTGGATCTTTAAAATGAATATTA	2064
	TAATATTCATTTTAAAGATCCAAGATATTACCATGTTGGTGGGA AGAGATATGACAAAACAGTAGAAACTGTCCAAGGTCCATCAA GAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGA	2065
	GTTTCTACTGTTTTGTC	2066
	GACAAAACAGTAGAAAC	2067
Haemophilia A Glu321Lys gGAA-AAA	ACAGCCTAATATAGCAAGACACTCTGACATTGTTTGGTTTGTC TGACTCCAGATGGCATGGAAGCTTATGTCAAAGTAGACAGCT GTCCAGAGGAACCCCAACTACGAATGAAAAATAATG	2068

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CATTATTTTTCATTCGTAGTTGGGGTTCCTCTGGACAGCTGTC TACTTTGACATAAGCTTCCATGCCATCTGGAGTCAGACAAACC AAACAATGTCAGAGTGTCTTGCTATATTAGGCTGT	2069
	ATGGCATGGAAGCTTAT	2070
	ATAAGCTTCCATGCCAT	2071
Haemophilia A Tyr323Term TATg-TAA	ATATAGCAAGACACTCTGACATTGTTTGGTTTGTCTGACTCCA GATGGCATGGAAGCTTATGTCAAAGTAGACAGCTGTCCAGAG GAACCCCAACTACGAATGAAAAATAATGAAGAAGCG	2072
	CGCTTCTTCATTATTTTTCATTCGTAGTTGGGGTTCCTCTGGA CAGCTGTCTACTTTGACATAAGCTTCCATGCCATCTGGAGTCA GACAAACCAAACAATGTCAGAGTGTCTTGCTATAT	2073
	GAAGCTTATGTCAAAGT	2074
	ACTTTGACATAAGCTTC	2075
Haemophilia A Val326Leu aGTA-CTA	AAGACACTCTGACATTGTTTGGTTTGTCTGACTCCAGATGGCA TGGAAGCTTATGTCAAAGTAGACAGCTGTCCAGAGGAACCCC AACTACGAATGAAAAATAATGAAGAAGCGGAAGACT	2076
	AGTCTTCCGCTTCTTCATTATTTTTCATTCGTAGTTGGGGTTC CTCTGGACAGCTGTCTACTTTGACATAAGCTTCCATGCCATCT GGAGTCAGACAAACCAAACAATGTCAGAGTGTCTT	2077
	ATGTCAAAGTAGACAGC	2078
	GCTGTCTACTTTGACAT	2079
Haemophilia A Cys329Arg cTGT-CGT	TGACATTGTTTGGTTTGTCTGACTCCAGATGGCATGGAAGCTT ATGTCAAAGTAGACAGCTGTCCAGAGGAACCCCAACTACGAA TGAAAAATAATGAAGAAGCGGAAGACTATGATGATG	2080
	CATCATCATAGTCTTCCGCTTCTTCATTATTTTTCATTCGTAGT TGGGGTTCCTCTGGACAGCTGTCTACTTTGACATAAGCTTCC ATGCCATCTGGAGTCAGACAAACCAAACAATGTCA	2081
	TAGACAGCTGTCCAGAG	2082
	CTCTGGACAGCTGTCTA	2083
Haemophilia A Cys329Tyr TGT-TAT	GACATTGTTTGGTTTGTCTGACTCCAGATGGCATGGAAGCTTA TGTCAAAGTAGACAGCTGTCCAGAGGAACCCCAACTACGAAT GAAAAATAATGAAGAAGCGGAAGACTATGATGATGA	2084
	TCATCATCATAGTCTTCCGCTTCTTCATTATTTTTCATTCGTAG TTGGGGTTCCTCTGGACAGCTGTCTACTTTGACATAAGCTTCC ATGCCATCTGGAGTCAGACAAACCAAACAATGTC	2085
	AGACAGCTGTCCAGAGG	2086
	CCTCTGGACAGCTGTCT	2087
Haemophilia A Arg336Term aCGA-TGA	ACTCCAGATGGCATGGAAGCTTATGTCAAAGTAGACAGCTGT CCAGAGGAACCCCAACTACGAATGAAAAATAATGAAGAAGCG GAAGACTATGATGATGATCTTACTGATTCTGAAATGG	2088

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCATTTCAGAATCAGTAAGATCATCATCATAGTCTTCCGCTTC TTCATTATTTTTCATTC**G**TAGTTGGGGTTCCTCTGGACAGCTG TCTACTTTGACATAAGCTTCCATGCCATCTGGAGT	2089
	CCCAACTA**C**GAATGAAA	2090
	TTTCATTC**G**TAGTTGGG	2091
Haemophilia A Arg372Cys tCGC-TGC	GATTCTGAAATGGATGTGGTCAGGTTTGATGATGACAACTCTC CTTCCTTTATCCAAATT**C**GCTCAGTTGCCAAGAAGCATCCTAA AACTTGGGTACATTACATTGCTGCTGAAGAGGAGG	2092
	CCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAGGATG CTTCTTGGCAACTGAGC**G**AATTTGGATAAAGGAAGGAGAGTT GTCATCATCAAACCTGACCACATCCATTTCAGAATC	2093
	TCCAAATT**C**GCTCAGTT	2094
	AACTGAGC**G**AATTTGGA	2095
Haemophilia A Arg372His CGC-CAC	ATTCTGAAATGGATGTGGTCAGGTTTGATGATGACAACTCTCC TTCCTTTATCCAAATTC**G**CTCAGTTGCCAAGAAGCATCCTAAA ACTTGGGTACATTACATTGCTGCTGAAGAGGAGGA	2096
	TCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAGGAT GCTTCTTGGCAACTGAG**C**GAATTTGGATAAAGGAAGGAGAGT TGTCATCATCAAACCTGACCACATCCATTTCAGAAT	2097
	CCAAATTC**G**CTCAGTTG	2098
	CAACTGAG**C**GAATTTGG	2099
Haemophilia A Ser373Leu TCA-TTA	CTGAAATGGATGTGGTCAGGTTTGATGATGACAACTCTCCTTC CTTTATCCAAATTCGCT**C**AGTTGCCAAGAAGCATCCTAAAACT TGGGTACATTACATTGCTGCTGAAGAGGAGGACTG	2100
	CAGTCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAG GATGCTTCTTGGCAACT**G**AGCGAATTTGGATAAAGGAAGGAG AGTTGTCATCATCAAACCTGACCACATCCATTTCAG	2101
	AATTCGCT**C**AGTTGCCA	2102
	TGGCAACT**G**AGCGAATT	2103
Haemophilia A Ser373Pro cTCA-CCA	TCTGAAATGGATGTGGTCAGGTTTGATGATGACAACTCTCCTT CCTTTATCCAAATTCGC**T**CAGTTGCCAAGAAGCATCCTAAAAC TTGGGTACATTACATTGCTGCTGAAGAGGAGGACT	2104
	AGTCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAGG ATGCTTCTTGGCAACTG**A**GCGAATTTGGATAAAGGAAGGAGA GTTGTCATCATCAAACCTGACCACATCCATTTCAGA	2105
	AAATTCGC**T**CAGTTGCC	2106
	GGCAACTG**A**GCGAATTT	2107
Haemophilia A Ser373Term TCA-TAA	CTGAAATGGATGTGGTCAGGTTTGATGATGACAACTCTCCTTC CTTTATCCAAATTCGCT**C**AGTTGCCAAGAAGCATCCTAAAACT TGGGTACATTACATTGCTGCTGAAGAGGAGGACTG	2108

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CAGTCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAG GATGCTTCTTGGCAACTGAGCGAATTTGGATAAAGGAAGGAG AGTTGTCATCATCAAACCTGACCACATCCATTTCAG	2109
	AATTCGCTCAGTTGCCA	2110
	TGGCAACTGAGCGAATT	2111
Haemophilia A Ile386Phe cATT-TTT	CCTTCCTTTATCCAAATTCGCTCAGTTGCCAAGAAGCATCCTA AAACTTGGGTACATTACATTGCTGCTGAAGAGGAGGACTGGG ACTATGCTCCCTTAGTCCTCGCCCCCGATGACAGGT	2112
	ACCTGTCATCGGGGGCGAGGACTAAGGGAGCATAGTCCCAG TCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAGGAT GCTTCTTGGCAACTGAGCGAATTTGGATAAAGGAAGG	2113
	TACATTACATTGCTGCT	2114
	AGCAGCAATGTAATGTA	2115
Haemophilia A Ile386Ser ATT-AGT	CTTCCTTTATCCAAATTCGCTCAGTTGCCAAGAAGCATCCTAA AACTTGGGTACATTACATTGCTGCTGAAGAGGAGGACTGGGA CTATGCTCCCTTAGTCCTCGCCCCCGATGACAGGTA	2116
	TACCTGTCATCGGGGGCGAGGACTAAGGGAGCATAGTCCCA GTCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAGGA TGCTTCTTGGCAACTGAGCGAATTTGGATAAAGGAAG	2117
	ACATTACATTGCTGCTG	2118
	CAGCAGCAATGTAATGT	2119
Haemophilia A Glu390Gly GAG-GGG	AAATTCGCTCAGTTGCCAAGAAGCATCCTAAAACTTGGGTACA TTACATTGCTGCTGAAGAGGAGGACTGGGACTATGCTCCCTT AGTCCTCGCCCCCGATGACAGGTAAGCACTTTTTGA	2120
	TCAAAAAGTGCTTACCTGTCATCGGGGGCGAGGACTAAGGGA GCATAGTCCCAGTCCTCCTCTTCAGCAGCAATGTAATGTACC CAAGTTTTAGGATGCTTCTTGGCAACTGAGCGAATTT	2121
	TGCTGAAGAGGAGGACT	2122
	AGTCCTCCTCTTCAGCA	2123
Haemophilia A Trp393Gly cTGG-GGG	TCAGTTGCCAAGAAGCATCCTAAAACTTGGGTACATTACATTG CTGCTGAAGAGGAGGACTGGGACTATGCTCCCTTAGTCCTCG CCCCCGATGACAGGTAAGCACTTTTTGACTATTGGT	2124
	ACCAATAGTCAAAAAGTGCTTACCTGTCATCGGGGGCGAGGA CTAAGGGAGCATAGTCCCAGTCCTCCTCTTCAGCAGCAATGT AATGTACCCAAGTTTTAGGATGCTTCTTGGCAACTGA	2125
	AGGAGGACTGGGACTAT	2126
	ATAGTCCCAGTCCTCCT	2127
Haemophilia A Lys408Ile AAA-ATA	GCCTACCTAGAATTTTTCTTCCCAACCTCTCATCTTTTTTTCTC TTATACAGAAGTTATAAAAGTCAATATTTGAACAATGGCCCTC AGCGGATTGGTAGGAAGTACAAAAAAGTCCGATT	2128

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AATCGGACTTTTTTGTACTTCCTACCAATCCGCTGAGGGCCAT TGTTCAAATATTGACTTTTATAACTTCTGTATAAGAGAAAAAAA GATGAGAGGTTGGGAAGAAAAATTCTAGGTAGGC	2129
	AAGTTATAAAAGTCAAT	2130
	ATTGACTTTTATAACTT	2131
Haemophilia A Leu412Phe TTGa-TTT	TTTTCTTCCCAACCTCTCATCTTTTTTTCTCTTATACAGAAGTT ATAAAAGTCAATATTTGAACAATGGCCCTCAGCGGATTGGTAG GAAGTACAAAAAAGTCCGATTTATGGCATACACA	2132
	TGTGTATGCCATAAATCGGACTTTTTTGTACTTCCTACCAATC CGCTGAGGGCCATTGTTCAAATATTGACTTTTATAACTTCTGT ATAAGAGAAAAAAAGATGAGAGGTTGGGAAGAAAA	2133
	CAATATTTGAACAATGG	2134
	CCATTGTTCAAATATTG	2135
Haemophilia A Arg418Trp gCGG-TGG	TCATCTTTTTTTCTCTTATACAGAAGTTATAAAAGTCAATATTTG AACAATGGCCCTCAGCGGATTGGTAGGAAGTACAAAAAAGTC CGATTTATGGCATACACAGATGAAACCTTTAAGA	2136
	TCTTAAAGGTTTCATCTGTGTATGCCATAAATCGGACTTTTTTG TACTTCCTACCAATCCGCTGAGGGCCATTGTTCAAATATTGAC TTTTATAACTTCTGTATAAGAGAAAAAAAGATGA	2137
	GCCCTCAGCGGATTGGT	2138
	ACCAATCCGCTGAGGGC	2139
Haemophilia A Gly420Val GGT-GTT	TTTTTCTCTTATACAGAAGTTATAAAAGTCAATATTTGAACAAT GGCCCTCAGCGGATTGGTAGGAAGTACAAAAAAGTCCGATTT ATGGCATACACAGATGAAACCTTTAAGACTCGTGA	2140
	TCACGAGTCTTAAAGGTTTCATCTGTGTATGCCATAAATCGGA CTTTTTTGTACTTCCTACCAATCCGCTGAGGGCCATTGTTCAA ATATTGACTTTTATAACTTCTGTATAAGAGAAAAA	2141
	GCGGATTGGGTAGGAAGT	2142
	ACTTCCTACCAATCCGC	2143
Haemophilia A Lys425Arg AAA-AGA	GAAGTTATAAAAGTCAATATTTGAACAATGGCCCTCAGCGGAT TGGTAGGAAGTACAAAAAAGTCCGATTTATGGCATACACAGAT GAAACCTTTAAGACTCGTGAAGCTATTCAGCATGA	2144
	TCATGCTGAATAGCTTCACGAGTCTTAAAGGTTTCATCTGTGT ATGCCATAAATCGGACTTTTTTGTACTTCCTACCAATCCGCTG AGGGCCATTGTTCAAATATTGACTTTTATAACTTC	2145
	GTACAAAAAAGTCCGAT	2146
	ATCGGACTTTTTTGTAC	2147
Haemophilia A Arg427Term cCGA-TGA	TATAAAAGTCAATATTTGAACAATGGCCCTCAGCGGATTGGTA GGAAGTACAAAAAAGTCCGATTTATGGCATACACAGATGAAAC CTTTAAGACTCGTGAAGCTATTCAGCATGAATCAG	2148

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTGATTCATGCTGAATAGCTTCACGAGTCTTAAAGGTTTCATC TGTGTATGCCATAAATC**G**GACTTTTTTGTACTTCCTACCAATC CGCTGAGGGCCATTGTTCAAATATTGACTTTTATA	2149
	AAAAAGTC**C**GATTTATG	2150
	CATAAATC**G**GACTTTTT	2151
Haemophilia A Tyr431Asn aTAC-AAC	TATTTGAACAATGGCCCTCAGCGGATTGGTAGGAAGTACAAA AAAGTCCGATTTATGGCA**T**ACACAGATGAAACCTTTAAGACTC GTGAAGCTATTCAGCATGAATCAGGAATCTTGGGAC	2152
	GTCCCAAGATTCCTGATTCATGCTGAATAGCTTCACGAGTCTT AAAGGTTTCATCTGTGT**A**TGCCATAAATCGGACTTTTTTGTAC TTCCTACCAATCCGCTGAGGGCCATTGTTCAAATA	2153
	TTATGGCA**T**ACACAGAT	2154
	ATCTGTGT**A**TGCCATAA	2155
Haemophilia A Thr435Ile ACC-ATC	GCCCTCAGCGGATTGGTAGGAAGTACAAAAAAGTCCGATTTA TGGCATACACAGATGAAA**C**CTTTAAGACTCGTGAAGCTATTCA GCATGAATCAGGAATCTTGGGACCTTTACTTTATGG	2156
	CCATAAAGTAAAGGTCCCAAGATTCCTGATTCATGCTGAATAG CTTCACGAGTCTTAAAG**G**TTTCATCTGTGTATGCCATAAATCG GACTTTTTTGTACTTCCTACCAATCCGCTGAGGGC	2157
	AGATGAAA**C**CTTTAAGA	2158
	TCTTAAAG**G**TTTCATCT	2159
Haemophilia A Pro451Leu CCT-CTT	ACACAGATGAAACCTTTAAGACTCGTGAAGCTATTCAGCATGA ATCAGGAATCTTGGGAC**C**TTTACTTTATGGGGAAGTTGGAGA CACACTGTTGGTAAGTTGAAGAAAAGATTTAAGGTC	2160
	GACCTTAAATCTTTTCTTCAACTTACCAACAGTGTGTCTCCAA CTTCCCCATAAAGTAAA**G**GTCCCAAGATTCCTGATTCATGCTG AATAGCTTCACGAGTCTTAAAGGTTTCATCTGTGT	2161
	CTTGGGAC**C**TTTACTTT	2162
	AAAGTAAA**G**GTCCCAAG	2163
Haemophilia A Pro451Thr aCCT-ACT	TACACAGATGAAACCTTTAAGACTCGTGAAGCTATTCAGCATG AATCAGGAATCTTGGGA**C**CTTTACTTTATGGGGAAGTTGGAGA CACACTGTTGGTAAGTTGAAGAAAAGATTTAAGGT	2164
	ACCTTAAATCTTTTCTTCAACTTACCAACAGTGTGTCTCCAACT TCCCCATAAAGTAAAG**G**TCCCAAGATTCCTGATTCATGCTGAA TAGCTTCACGAGTCTTAAAGGTTTCATCTGTGTA	2165
	TCTTGGGA**C**CTTTACTT	2166
	AAGTAAAG**G**TCCCAAGA	2167
Haemophilia A Gly455Arg tGGG-AGG	ACCTTTAAGACTCGTGAAGCTATTCAGCATGAATCAGGAATCT TGGGACCTTTACTTTAT**G**GGGAAGTTGGAGACACACTGTTGG TAAGTTGAAGAAAAGATTTAAGGTCAGGTAAGAAGA	2168

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCTTCTTACCTGACCTTAAATCTTTTCTTCAACTTACCAACAGT GTGTCTCCAACTTCCCCATAAAGTAAAGGTCCCAAGATTCCTG ATTCATGCTGAATAGCTTCACGAGTCTTAAAGGT	2169
	TACTTTATGGGGAAGTT	2170
	AACTTCCCCATAAAGTA	2171
Haemophilia A Gly455Glu GGG-GAG	CCTTTAAGACTCGTGAAGCTATTCAGCATGAATCAGGAATCTT GGGACCTTTACTTTATGGGGAAGTTGGAGACACACTGTTGGT AAGTTGAAGAAAAGATTTAAGGTCAGGTAAGAAGAA	2172
	TTCTTCTTACCTGACCTTAAATCTTTTCTTCAACTTACCAACAG TGTGTCTCCAACTTCCCCATAAAGTAAAGGTCCCAAGATTCCT GATTCATGCTGAATAGCTTCACGAGTCTTAAAGG	2173
	ACTTTATGGGGAAGTTG	2174
	CAACTTCCCCATAAAGT	2175
Haemophilia A Asp459Asn aGAC-AAC	CGTGAAGCTATTCAGCATGAATCAGGAATCTTGGGACCTTTAC TTTATGGGGAAGTTGGAGACACACTGTTGGTAAGTTGAAGAA AAGATTTAAGGTCAGGTAAGAAGAAAAAGTCTGGAG	2176
	CTCCAGACTTTTTCTTCTTACCTGACCTTAAATCTTTTCTTCAA CTTACCAACAGTGTGTCTCCAACTTCCCCATAAAGTAAAGGTC CCAAGATTCCTGATTCATGCTGAATAGCTTCACG	2177
	AAGTTGGAGACACACTG	2178
	CAGTGTGTCTCCAACTT	2179
Haemophilia A Phe465Cys TTT-TGT	TGTTGATCCTAGTCGTTTTAGGATTTGATCTTAGATCTCGCTTA TACTTTCAGATTATATTTTAAGAATCAAGCAAGCAGACCATATAA CATCTACCCTCACGGAATCACTGATGTCCGTCC	2180
	GGACGGACATCAGTGATTCCGTGAGGGTAGATGTTATATGGT CTGCTTGCTTGATTCTTAAATATAATCTGAAAGTATAAGCGAG ATCTAAGATCAAATCCTAAAACGACTAGGATCAACA	2181
	GATTATATTTTAAGAATC	2182
	GATTCTTAAATATAATC	2183
Haemophilia A Ala469Gly GCA-GGA	TCGTTTTAGGATTTGATCTTAGATCTCGCTTATACTTTCAGATT ATATTTAAGAATCAAGCAAGCAGACCATATAACATCTACCCTC ACGGAATCACTGATGTCCGTCCTTTGTATTCAAG	2184
	CTTGAATACAAAGGACGGACATCAGTGATTCCGTGAGGGTAG ATGTTATATGGTCTGCTTGCTTGATTCTTAAATATAATCTGAAA GTATAAGCGAGATCTAAGATCAAATCCTAAAACGA	2185
	GAATCAAGCAAGCAGAC	2186
	GTCTGCTTGCTTGATTC	2187
Haemophilia A Arg471Gly cAGA-GGA	TTAGGATTTGATCTTAGATCTCGCTTATACTTTCAGATTATATT TAAGAATCAAGCAAGCAGACCATATAACATCTACCCTCACGG AATCACTGATGTCCGTCCTTTGTATTCAAGGAGAT	2188

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATCTCCTTGAATACAAAGGACGGACATCAGTGATTCCGTGAG GGTAGATGTTATATGGTCTGCTTGCTTGATTCTTAAATATAATC TGAAAGTATAAGCGAGATCTAAGATCAAATCCTAA	2189
	AAGCAAGCAGACCATAT	2190
	ATATGGTCTGCTTGCTT	2191
Haemophilia A Tyr473Cys TAT-TGT	TTGATCTTAGATCTCGCTTATACTTTCAGATTATATTTAAGAAT CAAGCAAGCAGACCATATAACATCTACCCTCACGGAATCACT GATGTCCGTCCTTTGTATTCAAGGAGATTACCAAA	2192
	TTTGGTAATCTCCTTGAATACAAAGGACGGACATCAGTGATTC CGTGAGGGTAGATGTTATATGGTCTGCTTGCTTGATTCTTAAA TATAATCTGAAAGTATAAGCGAGATCTAAGATCAA	2193
	CAGACCATATAACATCT	2194
	AGATGTTATATGGTCTG	2195
Haemophilia A Tyr473His aTAT-CAT	TTTGATCTTAGATCTCGCTTATACTTTCAGATTATATTTAAGAA TCAAGCAAGCAGACCATATAACATCTACCCTCACGGAATCACT GATGTCCGTCCTTTGTATTCAAGGAGATTACCAA	2196
	TTGGTAATCTCCTTGAATACAAAGGACGGACATCAGTGATTCC GTGAGGGTAGATGTTATATGGTCTGCTTGCTTGATTCTTAAAT ATAATCTGAAAGTATAAGCGAGATCTAAGATCAAA	2197
	GCAGACCATATAACATC	2198
	GATGTTATATGGTCTGC	2199
Haemophilia A Ile475Thr ATC-ACC	TTAGATCTCGCTTATACTTTCAGATTATATTTAAGAATCAAGCA AGCAGACCATATAACATCTACCCTCACGGAATCACTGATGTCC GTCCTTTGTATTCAAGGAGATTACCAAAAGGTAA	2200
	TTACCTTTTGGTAATCTCCTTGAATACAAAGGACGGACATCAG TGATTCCGTGAGGGTAGATGTTATATGGTCTGCTTGCTTGATT CTTAAATATAATCTGAAAGTATAAGCGAGATCTAA	2201
	ATATAACATCTACCCTC	2202
	GAGGGTAGATGTTATAT	2203
Haemophilia A Gly479Arg cGGA-AGA	TTATACTTTCAGATTATATTTAAGAATCAAGCAAGCAGACCATA TAACATCTACCCTCACGGAATCACTGATGTCCGTCCTTTGTAT TCAAGGAGATTACCAAAAGGTAAATATTCCCTCG	2204
	CGAGGGAATATTTACCTTTTGGTAATCTCCTTGAATACAAAGG ACGGACATCAGTGATTCCGTGAGGGTAGATGTTATATGGTCT GCTTGCTTGATTCTTAAATATAATCTGAAAGTATAA	2205
	ACCCTCACGGAATCACT	2206
	AGTGATTCCGTGAGGGT	2207
Haemophilia A Thr522Ser aACT-TCT	CCAATTCTGCCAGGAGAAATATTCAAATATAAATGGACAGTGA CTGTAGAAGATGGGCCAACTAAATCAGATCCTCGGTGCCTGA CCCGCTATTACTCTAGTTTCGTTAATATGGAGAGAG	2208

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTCTCTCCATATTAACGAAACTAGAGTAATAGCGGGTCAGGC ACCGAGGATCTGATTTAGTTGGCCCATCTTCTACAGTCACTGT CCATTTATATTTGAATATTTCTCCTGGCAGAATTGG	2209
	ATGGGCCAAACTAAATCA	2210
	TGATTTAGTTGGCCCAT	2211
Haemophilia A Asp525Asn aGAT-AAT	CCAGGAGAAATATTCAAATATAAATGGACAGTGACTGTAGAAG ATGGGCCAACTAAATCAGATCCTCGGTGCCTGACCCGCTATT ACTCTAGTTTCGTTAATATGGAGAGAGATCTAGCTT	2212
	AAGCTAGATCTCTCTCCATATTAACGAAACTAGAGTAATAGCG GGTCAGGCACCGAGGATCTGATTTAGTTGGCCCATCTTCTAC AGTCACTGTCCATTTATATTTGAATATTTCTCCTGG	2213
	CTAAATCAGATCCTCGG	2214
	CCGAGGATCTGATTTAG	2215
Haemophilia A Arg527Trp tCGG-TGG	GAAATATTCAAATATAAATGGACAGTGACTGTAGAAGATGGGC CAACTAAATCAGATCCTCGGTGCCTGACCCGCTATTACTCTA GTTTCGTTAATATGGAGAGAGATCTAGCTTCAGGAC	2216
	GTCCTGAAGCTAGATCTCTCTCCATATTAACGAAACTAGAGTA ATAGCGGGTCAGGCACCGAGGATCTGATTTAGTTGGCCCATC TTCTACAGTCACTGTCCATTTATATTTGAATATTTC	2217
	CAGATCCTCGGTGCCTG	2218
	CAGGCACCGAGGATCTG	2219
Haemophilia A Arg531Cys cCGC-TGC	TATAAATGGACAGTGACTGTAGAAGATGGGCCAACTAAATCA GATCCTCGGTGCCTGACCCGCTATTACTCTAGTTTCGTTAATA TGGAGAGAGATCTAGCTTCAGGACTCATTGGCCCTC	2220
	GAGGGCCAATGAGTCCTGAAGCTAGATCTCTCTCCATATTAA CGAAACTAGAGTAATAGCGGGTCAGGCACCGAGGATCTGATT TAGTTGGCCCATCTTCTACAGTCACTGTCCATTTATA	2221
	GCCTGACCCGCTATTAC	2222
	GTAATAGCGGGTCAGGC	2223
Haemophilia A Arg531Gly cCGC-GGC	TATAAATGGACAGTGACTGTAGAAGATGGGCCAACTAAATCA GATCCTCGGTGCCTGACCCGCTATTACTCTAGTTTCGTTAATA TGGAGAGAGATCTAGCTTCAGGACTCATTGGCCCTC	2224
	GAGGGCCAATGAGTCCTGAAGCTAGATCTCTCTCCATATTAA CGAAACTAGAGTAATAGCGGGTCAGGCACCGAGGATCTGATT TAGTTGGCCCATCTTCTACAGTCACTGTCCATTTATA	2225
	GCCTGACCCGCTATTAC	2226
	GTAATAGCGGGTCAGGC	2227
Haemophilia A Arg531His CGC-CAC	ATAAATGGACAGTGACTGTAGAAGATGGGCCAACTAAATCAG ATCCTCGGTGCCTGACCCGCTATTACTCTAGTTTCGTTAATAT GGAGAGAGATCTAGCTTCAGGACTCATTGGCCCTCT	2228

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGAGGGCCAATGAGTCCTGAAGCTAGATCTCTCTCCATATTAA CGAAACTAGAGTAATAGCGGGTCAGGCACCGAGGATCTGATT TAGTTGGCCCATCTTCTACAGTCACTGTCCATTTAT	2229
	CCTGACCCGCTATTACT	2230
	AGTAATAGCGGGTCAGG	2231
Haemophilia A Ser534Pro cTCT-CCT	ACAGTGACTGTAGAAGATGGGCCAACTAAATCAGATCCTCGG TGCCTGACCCGCTATTACTCTAGTTTCGTTAATATGGAGAGAG ATCTAGCTTCAGGACTCATTGGCCCTCTCCTCATCT	2232
	AGATGAGGAGAGGGCCAATGAGTCCTGAAGCTAGATCTCTCT CCATATTAACGAAACTAGAGTAATAGCGGGTCAGGCACCGAG GATCTGATTTAGTTGGCCCATCTTCTACAGTCACTGT	2233
	GCTATTACTCTAGTTTC	2234
	GAAACTAGAGTAATAGC	2235
Haemophilia A Ser535Gly tAGT-GGT	GTGACTGTAGAAGATGGGCCAACTAAATCAGATCCTCGGTGC CTGACCCGCTATTACTCTAGTTTCGTTAATATGGAGAGAGATC TAGCTTCAGGACTCATTGGCCCTCTCCTCATCTGCT	2236
	AGCAGATGAGGAGAGGGCCAATGAGTCCTGAAGCTAGATCTC TCTCCATATTAACGAAACTAGAGTAATAGCGGGTCAGGCACC GAGGATCTGATTTAGTTGGCCCATCTTCTACAGTCAC	2237
	ATTACTCTAGTTTCGTT	2238
	AACGAAACTAGAGTAAT	2239
Haemophilia A Val537Asp GTT-GAT	TAGAAGATGGGCCAACTAAATCAGATCCTCGGTGCCTGACCC GCTATTACTCTAGTTTCGTTAATATGGAGAGAGATCTAGCTTC AGGACTCATTGGCCCTCTCCTCATCTGCTACAAAGA	2240
	TCTTTGTAGCAGATGAGGAGAGGGCCAATGAGTCCTGAAGCT AGATCTCTCTCCATATTAACGAAACTAGAGTAATAGCGGGTCA GGCACCGAGGATCTGATTTAGTTGGCCCATCTTCTA	2241
	TAGTTTCGTTAATATGG	2242
	CCATATTAACGAAACTA	2243
Haemophilia A Arg541Thr AGA-ACA	CAACTAAATCAGATCCTCGGTGCCTGACCCGCTATTACTCTA GTTTCGTTAATATGGAGAGAGATCTAGCTTCAGGACTCATTGG CCCTCTCCTCATCTGCTACAAAGAATCTGTAGATCA	2244
	TGATCTACAGATTCTTTGTAGCAGATGAGGAGAGGGCCAATG AGTCCTGAAGCTAGATCTCTCTCCATATTAACGAAACTAGAGT AATAGCGGGTCAGGCACCGAGGATCTGATTTAGTTG	2245
	TATGGAGAGAGATCTAG	2246
	CTAGATCTCTCTCCATA	2247
Haemophilia A Asp542Gly GAT-GGT	CTAAATCAGATCCTCGGTGCCTGACCCGCTATTACTCTAGTTT CGTTAATATGGAGAGAGATCTAGCTTCAGGACTCATTGGCCC TCTCCTCATCTGCTACAAAGAATCTGTAGATCAAAG	2248

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTTTGATCTACAGATTCTTTGTAGCAGATGAGGAGAGGGCCA ATGAGTCCTGAAGCTAGATCTCTCTCCATATTAACGAAACTAG AGTAATAGCGGGTCAGGCACCGAGGATCTGATTTAG	2249
	GGAGAGAGATCTAGCTT	2250
	AAGCTAGATCTCTCTCC	2251
Haemophilia A Asp542His aGAT-CAT	ACTAAATCAGATCCTCGGTGCCTGACCCGCTATTACTCTAGTT TCGTTAATATGGAGAGAGATCTAGCTTCAGGACTCATTGGCC CTCTCCTCATCTGCTACAAAGAATCTGTAGATCAAA	2252
	TTTGATCTACAGATTCTTTGTAGCAGATGAGGAGAGGGCCAAT GAGTCCTGAAGCTAGATCTCTCTCCATATTAACGAAACTAGAG TAATAGCGGGTCAGGCACCGAGGATCTGATTTAGT	2253
	TGGAGAGAGATCTAGCT	2254
	AGCTAGATCTCTCTCCA	2255
Haemophilia A Asp542Tyr aGAT-TAT	ACTAAATCAGATCCTCGGTGCCTGACCCGCTATTACTCTAGTT TCGTTAATATGGAGAGAGATCTAGCTTCAGGACTCATTGGCC CTCTCCTCATCTGCTACAAAGAATCTGTAGATCAAA	2256
	TTTGATCTACAGATTCTTTGTAGCAGATGAGGAGAGGGCCAAT GAGTCCTGAAGCTAGATCTCTCTCCATATTAACGAAACTAGAG TAATAGCGGGTCAGGCACCGAGGATCTGATTTAGT	2257
	TGGAGAGAGATCTAGCT	2258
	AGCTAGATCTCTCTCCA	2259
Haemophilia A Glu557Term aGAA-TAA	GTTAATATGGAGAGAGATCTAGCTTCAGGACTCATTGGCCCT CTCCTCATCTGCTACAAAGAATCTGTAGATCAAAGAGGAAACC AGGTGAGTTCTTGCCTTTCCAAGTGCTGGGTTTCAT	2260
	ATGAAACCCAGCACTTGGAAAGGCAAGAACTCACCTGGTTTC CTCTTTGATCTACAGATTCTTTGTAGCAGATGAGGAGAGGGC CAATGAGTCCTGAAGCTAGATCTCTCTCCATATTAAC	2261
	GCTACAAAGAATCTGTA	2262
	TACAGATTCTTTGTAGC	2263
Haemophilia A Ser558Phe TCT-TTT	ATATGGAGAGAGATCTAGCTTCAGGACTCATTGGCCCTCTCC TCATCTGCTACAAAGAATCTGTAGATCAAAGAGGAAACCAGGT GAGTTCTTGCCTTTCCAAGTGCTGGGTTTCATTCTC	2264
	GAGAATGAAACCCAGCACTTGGAAAGGCAAGAACTCACCTGG TTTCCTCTTTGATCTACAGATTCTTTGTAGCAGATGAGGAGAG GGCCAATGAGTCCTGAAGCTAGATCTCTCTCCATAT	2265
	CAAAGAATCTGTAGATC	2266
	GATCTACAGATTCTTTG	2267
Haemophilia A Val559Ala GTA-GCA	TGGAGAGAGATCTAGCTTCAGGACTCATTGGCCCTCTCCTCA TCTGCTACAAAGAATCTGTAGATCAAAGAGGAAACCAGGTGA GTTCTTGCCTTTCCAAGTGCTGGGTTTCATTCTCAGT	2268

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACTGAGAATGAAACCCAGCACTTGGAAAGGCAAGAACTCACC TGGTTTCCTCTTTGATCTACAGATTCTTTGTAGCAGATGAGGA GAGGGCCAATGAGTCCTGAAGCTAGATCTCTCTCCA	2269
	AGAATCTGTAGATCAAA	2270
	TTTGATCTACAGATTCT	2271

EXAMPLE 14
Hemophilia - Factor IX Deficiency

The attached table discloses the correcting oligonucleotide base sequences for the Factor IX oligonucleotides of the invention.

Table 21
Factor IX Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia B Asn2Asp tAAT-GAT	ATTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAA TCGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTT CAAGGGAACCTTGAGAGAGAATGTATGGAAGAAA	2272
	TTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTCT TCCAATTTACCTGAATTATACCTCTTTGGCCGATTCAGAATTTT GTTGGCGTTTTCATGATCAAGAAAAACTGAAAT	2273
	AGAGGTATAATTCAGGT	2274
	ACCTGAATTATACCTCT	2275
Haemophilia B Asn2Ile AAT-ATT	TTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAAT CGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTT CAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAA	2276
	TTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTC TTCCAATTTACCTGAATTATACCTCTTTGGCCGATTCAGAATTT TGTTGGCGTTTTCATGATCAAGAAAAACTGAAA	2277
	GAGGTATAATTCAGGTA	2278
	TACCTGAATTATACCTC	2279
Haemophilia B Asn2Tyr tAAT-TAT	ATTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAA TCGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTT CAAGGGAACCTTGAGAGAGAATGTATGGAAGAAA	2280
	TTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTCT TCCAATTTACCTGAATTATACCTCTTTGGCCGATTCAGAATTTT GTTGGCGTTTTCATGATCAAGAAAAACTGAAAT	2281

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGAGGTATAATTCAGGT	2282
	ACCTGAATTATACCTCT	2283
Haemophilia B Ser3Pro tTCA-CCA	TCAGTTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAATC GGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCA AGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGT	2284
	ACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAAC TCTTCCAATTTACCTGAATTATACCTCTTTGGCCGATTCAGAA TTTTGTTGGCGTTTTCATGATCAAGAAAAACTGA	2285
	GGTATAATTCAGGTAAA	2286
	TTTACCTGAATTATACC	2287
Haemophilia B Gly4Asp GGT-GAT	TTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAATCGGCC AAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGG AACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAG	2288
	CTACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAAC AAACTCTTCCAATTTACCTGAATTATACCTCTTTGGCCGATTCA GAATTTTGTTGGCGTTTTCATGATCAAGAAAAA	2289
	TAATTCAGGTAAATTGG	2290
	CCAATTTACCTGAATTA	2291
Haemophilia B Gly4Ser aGGT-AGT	GTTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAATCGGC CAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGG GAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTA	2292
	TACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACA AACTCTTCCAATTTACCTGAATTATACCTCTTTGGCCGATTCA GAATTTTGTTGGCGTTTTCATGATCAAGAAAAAC	2293
	ATAATTCAGGTAAATTG	2294
	CAATTTACCTGAATTAT	2295
Haemophilia B Lys5Glu tAAA-GAA	TTTCTTGATCATGAAAACGCCAACAAAATTCTGAATCGGCCAA AGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAA CCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTT	2296
	AACTACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGA ACAAACTCTTCCAATTTACCTGAATTATACCTCTTTGGCCGATT CAGAATTTTGTTGGCGTTTTCATGATCAAGAAA	2297
	ATTCAGGTAAATTGGAA	2298
	TTCCAATTTACCTGAAT	2299
Haemophilia B Glu7Ala GAA-GCA	ATCATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGTA TAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAG AGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGA	2300
	TCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAGGTT CCCTTGAACAAACTCTTCCAATTTACCTGAATTATACCTCTTTG GCCGATTCAGAATTTTGTTGGCGTTTTCATGAT	2301

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAAATTGGAAGAGTTTG	2302
	CAAACTCTTCCAATTTA	2303
Haemophilia B Glu7Lys gGAA-AAA	GATCATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGG TATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTG AGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAG	2304
	CTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAGGTTC CCTTGAACAAACTCTTCCAATTTACCTGAATTATACCTCTTTGG CCGATTCAGAATTTTGTTGGCGTTTTCATGATC	2305
	GTAAATTGGAAGAGTTT	2306
	AAACTCTTCCAATTTAC	2307
Haemophilia B Glu7Val GAA-GTA	ATCATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGTA TAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAG AGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGA	2308
	TCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAGGTT CCCTTGAACAAACTCTTCCAATTTACCTGAATTATACCTCTTTG GCCGATTCAGAATTTTGTTGGCGTTTTCATGAT	2309
	TAAATTGGAAGAGTTTG	2310
	CAAACTCTTCCAATTTA	2311
Haemophilia B Glu8Ala GAG-GCG	ATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGTATAA TTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAG AGAATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGC	2312
	GCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAG GTTCCCTTGAACAAACTCTTCCAATTTACCTGAATTATACCTCT TTGGCCGATTCAGAATTTTGTTGGCGTTTTCAT	2313
	ATTGGAAGAGTTTGTTC	2314
	GAACAAACTCTTCCAAT	2315
Haemophilia B Glu8Gly GAG-GGG	ATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGTATAA TTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAG AGAATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGC	2316
	GCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAG GTTCCCTTGAACAAACTCTTCCAATTTACCTGAATTATACCTCT TTGGCCGATTCAGAATTTTGTTGGCGTTTTCAT	2317
	ATTGGAAGAGTTTGTTC	2318
	GAACAAACTCTTCCAAT	2319
Haemophilia B Phe9Cys TTT-TGT	AAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGTATAATTC AGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGA ATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGCACG	2320
	CGTGCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTC AAGGTTCCCTTGAACAAACTCTTCCAATTTACCTGAATTATAC CTCTTTGGCCGATTCAGAATTTTGTTGGCGTTTT	2321

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GGAAGAGTTTGTTCAAG	2322
	CTTGAACAAACTCTTCC	2323
Haemophilia B Phe9Ile gTTT-ATT	GAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGTATAATT CAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAG AATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGCAC	2324
	GTGCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCA AGGTTCCCTTGAACAAACTCTTCCAATTTACCTGAATTATACC TCTTTGGCCGATTCAGAATTTTGTTGGCGTTTTC	2325
	TGGAAGAGTTTGTTCAA	2326
	TTGAACAAACTCTTCCA	2327
Haemophilia B Arg(-1)Ser AGGt-AGC	TTACATTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAATTC TGAATCGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTT TGTTCAAGGGAACCTTGAGAGAGAATGTATGGAA	2328
	TTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTCTTCC AATTTACCTGAATTATACCTCTTTGGCCGATTCAGAATTTTGTT GGCGTTTTCATGATCAAGAAAAACTGAAATGTAA	2329
	CCAAAGAGGTATAATTC	2330
	GAATTATACCTCTTTGG	2331
Haemophilia B Arg(-1)Thr AGG-ACG	TTTACATTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAATT CTGAATCGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAG TTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGA	2332
	TCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCA ATTTACCTGAATTATACCTCTTTGGCCGATTCAGAATTTTGTTG GCGTTTTCATGATCAAGAAAAACTGAAATGTAAA	2333
	GCCAAAGAGGTATAATT	2334
	AATTATACCTCTTTGGC	2335
Haemophilia B Lys(-2)Asn AAGa-AAT	CTTTTACATTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAA TTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGA GTTTGTTCAAGGGAACCTTGAGAGAGAATGTATG	2336
	CATACATTCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAAT TTACCTGAATTATACCTCTTTGGCCGATTCAGAATTTTGTTGG CGTTTTCATGATCAAGAAAAACTGAAATGTAAAAG	2337
	CGGCCAAAGAGGTATAA	2338
	TTATACCTCTTTGGCCG	2339
Haemophilia B Arg(-4)Gln CGG-CAG	AATTATTCTTTTACATTTCAGTTTTTCTTGATCATGAAAACGCC AACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAAT TGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGA	2340
	TCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCTG AATTATACCTCTTTGGCCGATTCAGAATTTTGTTGGCGTTTTCA TGATCAAGAAAAACTGAAATGTAAAAGAATAATT	2341

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCTGAATCGGCCAAAGA	2342
	TCTTTGGCCGATTCAGA	2343
Haemophilia B Arg(-4)Leu CGG-CTG	AATTATTCTTTTACATTTCAGTTTTTCTTGATCATGAAAACGCC AACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAAT TGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGA	2344
	TCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCTG AATTATACCTCTTTGGCCGATTCAGAATTTTGTTGGCGTTTTCA TGATCAAGAAAAACTGAAATGTAAAAGAATAATT	2345
	TCTGAATCGGCCAAAGA	2346
	TCTTTGGCCGATTCAGA	2347
Haemophilia B Arg(-4)Trp tCGG-TGG	GAATTATTCTTTTACATTTCAGTTTTTCTTGATCATGAAAACGC CAACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAA TTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAG	2348
	CTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCTGA ATTATACCTCTTTGGCCGATTCAGAATTTTGTTGGCGTTTTCAT GATCAAGAAAAACTGAAATGTAAAAGAATAATTC	2349
	TTCTGAATCGGCCAAAG	2350
	CTTTGGCCGATTCAGAA	2351
Haemophilia B Gln11Term tCAA-TAA	GCCAACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTA AATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTAT GGAAGAAAAGTGTAGTTTTGAAGAAGCACGAGAAG	2352
	CTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACATTCT CTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCTGAAT TATACCTCTTTGGCCGATTCAGAATTTTGTTGGC	2353
	AGTTTGTTCAAGGGAAC	2354
	GTTCCCTTGAACAAACT	2355
Haemophilia B Gly12Ala GGG-GCG	ACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAATT GGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGA AGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTT	2356
	AAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACA TTCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCT GAATTATACCTCTTTGGCCGATTCAGAATTTTGT	2357
	TGTTCAAGGGAACCTTG	2358
	CAAGGTTCCCTTGAACA	2359
Haemophilia B Gly12Arg aGGG-AGG	AACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAAT TGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGG AAGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTT	2360
	AAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACAT TCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCTG AATTATACCTCTTTGGCCGATTCAGAATTTTGTT	2361

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTGTTCAAGGGAACCTT	2362
	AAGGTTCCCTTGAACAA	2363
Haemophilia B Gly12Glu GGG-GAG	ACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAATT GGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGA AGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTT	2364
	AAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACA TTCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCT GAATTATACCTCTTTGGCCGATTCAGAATTTTGT	2365
	TGTTCAAGGGAACCTTG	2366
	CAAGGTTCCCTTGAACA	2367
Haemophilia B Glu17Gln aGAA-CAA	CGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTC AAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTT TTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAA	2368
	TTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACAC TTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTC TTCCAATTTACCTGAATTATACCTCTTTGGCCG	2369
	TTGAGAGAGAATGTATG	2370
	CATACATTCTCTCTCAA	2371
Haemophilia B Glu17Lys aGAA-AAA	CGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTC AAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTT TTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAA	2372
	TTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACAC TTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTC TTCCAATTTACCTGAATTATACCTCTTTGGCCG	2373
	TTGAGAGAGAATGTATG	2374
	CATACATTCTCTCTCAA	2375
Haemophilia B Cys18Arg aTGT-CGT	CCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAG GGAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTTTTG AAGAAGCACGAGAAGTTTTTGAAAACACTGAAAGAA	2376
	TTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTA CACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAA ACTCTTCCAATTTACCTGAATTATACCTCTTTGG	2377
	AGAGAGAATGTATGGAA	2378
	TTCCATACATTCTCTCT	2379
Haemophilia B Cys18Tyr TGT-TAT	CAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGG GAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAA GAAGCACGAGAAGTTTTTGAAAACACTGAAAGAAC	2380
	GTTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACT ACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAA ACTCTTCCAATTTACCTGAATTATACCTCTTTG	2381

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAGAGAATGTATGGAAG	2382
	CTTCCATACATTCTCTC	2383
Haemophilia B Glu20Val GAA-GTA	GGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCT TGAGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGC ACGAGAAGTTTTTGAAAACACTGAAAGAACAGTGAG	2384
	CTCACTGTTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTC AAAACTACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTT GAACAAACTCTTCCAATTTACCTGAATTATACC	2385
	ATGTATGGAAGAAAAGT	2386
	ACTTTTCTTCCATACAT	2387
Haemophilia B Glu21Lys aGAA-AAA	TATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTG AGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGCAC GAGAAGTTTTTGAAAACACTGAAAGAACAGTGAGTA	2388
	TACTCACTGTTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCT TCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCC TTGAACAAACTCTTCCAATTTACCTGAATTATA	2389
	GTATGGAAGAAAAGTGT	2390
	ACACTTTTCTTCCATAC	2391
Haemophilia B Cys23Arg gTGT-CGT	TCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGA GAATGTATGGAAGAAAAGTTGTAGTTTTGAAGAAGCACGAGAA GTTTTTGAAAACACTGAAAGAACAGTGAGTATTTCCA	2392
	TGGAAATACTCACTGTTCTTTCAGTGTTTTCAAAAACTTCTCGT GCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAG GTTCCCTTGAACAAACTCTTCCAATTTACCTGA	2393
	AAGAAAAGTTGTAGTTTT	2394
	AAAACTACACTTTTCTT	2395
Haemophilia B Cys23Tyr TGT-TAT	CAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAG AATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGT TTTTGAAAACACTGAAAGAACAGTGAGTATTTCCAC	2396
	GTGGAAATACTCACTGTTCTTTCAGTGTTTTCAAAAACTTCTC GTGCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCA AGGTTCCCTTGAACAAACTCTTCCAATTTACCTG	2397
	AGAAAAGTGTAGTTTTG	2398
	CAAAACTACACTTTTCT	2399
Haemophilia B Phe25Ser TTT-TCT	AATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTAT GGAAGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAA AACACTGAAAGAACAGTGAGTATTTCCACATAATA	2400
	TATTATGTGGAAATACTCACTGTTCTTTCAGTGTTTTCAAAAAC TTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACATTCTC TCTCAAGGTTCCCTTGAACAAACTCTTCCAATT	2401

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GTGTAGTTTTGAAGAAG	2402
	CTTCTTCAAAACTACAC	2403
Haemophilia B Glu26Gln tGAA-CAA	TTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATG GAAGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAA ACACTGAAAGAACAGTGAGTATTTCCACATAATACC	2404
	GGTATTATGTGGAAATACTCACTGTTCTTTCAGTGTTTTCAAAA ACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACATTC TCTCTCAAGGTTCCCTTGAACAAACTCTTCCAA	2405
	GTAGTTTTGAAGAAGCA	2406
	TGCTTCTTCAAAACTAC	2407
Haemophilia B Glu27Ala GAA-GCA	AAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAG AAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACAC TGAAAGAACAGTGAGTATTTCCACATAATACCCTTC	2408
	GAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAGTGTTTT CAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATA CATTCTCTCTCAAGGTTCCCTTGAACAAACTCTT	2409
	TTTTGAAGAAGCACGAG	2410
	CTCGTGCTTCTTCAAAA	2411
Haemophilia B Glu27Asp GAAg-GAC	AGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGA AAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACT GAAAGAACAGTGAGTATTTCCACATAATACCCTTCA	2412
	TGAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAGTGTTT TCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCAT ACATTCTCTCTCAAGGTTCCCTTGAACAAACTCT	2413
	TTTGAAGAAGCACGAGA	2414
	TCTCGTGCTTCTTCAAA	2415
Haemophilia B Glu27Lys aGAA-AAA	GAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAA GAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACA CTGAAAGAACAGTGAGTATTTCCACATAATACCCTT	2416
	AAGGGTATTATGTGGAAATACTCACTGTTCTTTCAGTGTTTTC AAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATAC ATTCTCTCTCAAGGTTCCCTTGAACAAACTCTTC	2417
	GTTTTGAAGAAGCACGA	2418
	TCGTGCTTCTTCAAAAC	2419
Haemophilia B Glu27Val GAA-GTA	AAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAG AAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACAC TGAAAGAACAGTGAGTATTTCCACATAATACCCTTC	2420
	GAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAGTGTTTT CAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATA CATTCTCTCTCAAGGTTCCCTTGAACAAACTCTT	2421

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTTTGAAGAAGCACGAG	2422
	CTCGTGCTTCTTCAAAA	2423
Haemophilia B Arg29Gln CGA-CAA	TTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGT GTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAAG AACAGTGAGTATTTCCACATAATACCCTTCAGATGC	2424
	GCATCTGAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAG TGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCT TCCATACATTCTCTCTCAAGGTTCCCTTGAACAA	2425
	AGAAGCACGAGAAGTTT	2426
	AAACTTCTCGTGCTTCT	2427
Haemophilia B Arg29Pro CGA-CCA	TTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGT GTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAAG AACAGTGAGTATTTCCACATAATACCCTTCAGATGC	2428
	GCATCTGAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAG TGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCT TCCATACATTCTCTCTCAAGGTTCCCTTGAACAA	2429
	AGAAGCACGAGAAGTTT	2430
	AAACTTCTCGTGCTTCT	2431
Haemophilia B Arg29Term aCGA-TGA	TTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGT GTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAAG AACAGTGAGTATTTCCACATAATACCCTTCAGATG	2432
	CATCTGAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAGT GTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTT CCATACATTCTCTCTCAAGGTTCCCTTGAACAAA	2433
	AAGAAGCACGAGAAGTT	2434
	AACTTCTCGTGCTTCTT	2435
Haemophilia B Glu30Lys aGAA-AAA	GTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGTGT AGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAAGAA CAGTGAGTATTTCCACATAATACCCTTCAGATGCAG	2436
	CTGCATCTGAAGGGTATTATGTGGAAATACTCACTGTTCTTTC AGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTT CTTCCATACATTCTCTCTCAAGGTTCCCTTGAAC	2437
	AAGCACGAGAAGTTTTT	2438
	AAAAACTTCTCGTGCTT	2439
Haemophilia B Glu30Term aGAA-TAA	GTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGTGT AGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAAGAA CAGTGAGTATTTCCACATAATACCCTTCAGATGCAG	2440
	CTGCATCTGAAGGGTATTATGTGGAAATACTCACTGTTCTTTC AGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTT CTTCCATACATTCTCTCTCAAGGTTCCCTTGAAC	2441

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AAGCACGAGAAGTTTTT	2442
	AAAAACTTCTCGTGCTT	2443
Haemophilia B Glu33Asp GAAa-GAC	CCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGAA GCACGAGAAGTTTTTGAAAACACTGAAAGAACAGTGAGTATTT CCACATAATACCCTTCAGATGCAGAGCATAGAATA	2444
	TATTCTATGCTCTGCATCTGAAGGGTATTATGTGGAAATACTC ACTGTTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAA ACTACACTTTTCTTCCATACATTCTCTCTCAAGG	2445
	GTTTTTGAAAACACTGA	2446
	TCAGTGTTTTCAAAAAC	2447
Haemophilia B Glu33Term tGAA-TAA	AACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAG AAGCACGAGAAGTTTTTGAAAACACTGAAAGAACAGTGAGTAT TTCCACATAATACCCTTCAGATGCAGAGCATAGAA	2448
	TTCTATGCTCTGCATCTGAAGGGTATTATGTGGAAATACTCAC TGTTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAAC TACACTTTTCTTCCATACATTCTCTCTCAAGGTT	2449
	AAGTTTTTGAAAACACT	2450
	AGTGTTTTCAAAAACTT	2451
Haemophilia B Trp42Term TGG-TAG	CAAAACACTTTAGATATTACCGTTAATTTGTCTTCTTTTATTCTT TATAGACTGAATTTTGGAAGCAGTATGTTGGTAAGCAATTCAT TTTATCCTCTAGCTAATATATGAAACATATGAG	2452
	CTCATATGTTTCATATATTAGCTAGAGGATAAAATGAATTGCTT ACCAACATACTGCTTCCAAAATTCAGTCTATAAAGAATAAAAG AAGACAAATTAACGGTAATATCTAAAGTGTTTTG	2453
	TGAATTTTGGAAGCAGT	2454
	ACTGCTTCCAAAATTCA	2455
Haemophilia B Lys43Glu gAAG-GAG	AAACACTTTAGATATTACCGTTAATTTGTCTTCTTTTATTCTTTA TAGACTGAATTTTGGAAGCAGTATGTTGGTAAGCAATTCATTT TATCCTCTAGCTAATATATGAAACATATGAGAA	2456
	TTCTCATATGTTTCATATATTAGCTAGAGGATAAAATGAATTGC TTACCAACATACTGCTTCCAAAATTCAGTCTATAAAGAATAAAA GAAGACAAATTAACGGTAATATCTAAAGTGTTT	2457
	AATTTTGGAAGCAGTAT	2458
	ATACTGCTTCCAAAATT	2459
Haemophilia B Gln44Term gCAG-TAG	CACTTTAGATATTACCGTTAATTTGTCTTCTTTTATTCTTTATAG ACTGAATTTTGGAAGCAGTATGTTGGTAAGCAATTCATTTTATC CTCTAGCTAATATATGAAACATATGAGAATTA	2460
	TAATTCTCATATGTTTCATATATTAGCTAGAGGATAAAATGAAT TGCTTACCAACATACTGCTTCCAAAATTCAGTCTATAAAGAATA AAAGAAGACAAATTAACGGTAATATCTAAAGTG	2461

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTTGGAAGCAGTATGTT	2462
	AACATACTGCTTCCAAA	2463
Haemophilia B Asp49Gly GAT-GGT	CCGGGCATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAAC CTATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTA AATGGCGGCAGTTGCAAGGATGACATTAATTCCTA	2464
	TAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAACATG GATTGGACTCACACTGATCTCCATCTTTGAGATAGGTTAAGAA ATTGAATTGGCACGTAAACTGCTTAGAATGCCCGG	2465
	AGATGGAGATCAGTGTG	2466
	CACACTGATCTCCATCT	2467
Haemophilia B Gln50His CAGt-CAC	GCATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTATC TCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGG CGGCAGTTGCAAGGATGACATTAATTCCTATGAA	2468
	TTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAA CATGGATTGGACTCACACTGATCTCCATCTTTGAGATAGGTTA AGAAATTGAATTGGCACGTAAACTGCTTAGAATGC	2469
	GGAGATCAGTGTGAGTC	2470
	GACTCACACTGATCTCC	2471
Haemophilia B Gln50Pro CAG-CCG	GGCATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTA TCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAAT GGCGGCAGTTGCAAGGATGACATTAATTCCTATGA	2472
	TCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAAC ATGGATTGGACTCACACTGATCTCCATCTTTGAGATAGGTTAA GAAATTGAATTGGCACGTAAACTGCTTAGAATGCC	2473
	TGGAGATCAGTGTGAGT	2474
	ACTCACACTGATCTCCA	2475
Haemophilia B Gln50Term tCAG-TAG	GGGCATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCT ATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAA TGGCGGCAGTTGCAAGGATGACATTAATTCCTATG	2476
	CATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAACA TGGATTGGACTCACACTGATCTCCATCTTTGAGATAGGTTAAG AAATTGAATTGGCACGTAAACTGCTTAGAATGCCC	2477
	ATGGAGATCAGTGTGAG	2478
	CTCACACTGATCTCCAT	2479
Haemophilia B Cys51Arg gTGT-CGT	CATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTATCT CAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGG CGGCAGTTGCAAGGATGACATTAATTCCTATGAAT	2480
	ATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAA ACATGGATTGGACTCACACTGATCTCCATCTTTGAGATAGGTT AAGAAATTGAATTGGCACGTAAACTGCTTAGAATG	2481

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAGATCAGTGTGAGTCC	2482
	GGACTCACACTGATCTC	2483
Haemophilia B Cys51Ser gTGT-AGT	CATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTATCT CAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGG CGGCAGTTGCAAGGATGACATTAATTCCTATGAAT	2484
	ATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAA ACATGGATTGGACTCACACTGATCTCCATCTTTGAGATAGGTT AAGAAATTGAATTGGCACGTAAACTGCTTAGAATG	2485
	GAGATCAGTGTGAGTCC	2486
	GGACTCACACTGATCTC	2487
Haemophilia B Cys51Trp TGTg-TGG	TTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTATCTCA AAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGGCG GCAGTTGCAAGGATGACATTAATTCCTATGAATGT	2488
	ACATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTT AAACATGGATTGGACTCACACTGATCTCCATCTTTGAGATAGG TTAAGAAATTGAATTGGCACGTAAACTGCTTAGAA	2489
	GATCAGTGTGAGTCCAA	2490
	TTGGACTCACACTGATC	2491
Haemophilia B Glu52Term tGAG-TAG	TCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTATCTCAA AGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGGCGG CAGTTGCAAGGATGACATTAATTCCTATGAATGTT	2492
	AACATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATT TAAACATGGATTGGACTCACACTGATCTCCATCTTTGAGATAG GTTAAGAAATTGAATTGGCACGTAAACTGCTTAGA	2493
	ATCAGTGTGAGTCCAAT	2494
	ATTGGACTCACACTGAT	2495
Haemophilia B Pro55Ala tCCA-GCA	TTTACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAG ATCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCA AGGATGACATTAATTCCTATGAATGTTGGTGTCCCT	2496
	AGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAACT GCCGCCATTTAAACATGGATTGGACTCACACTGATCTCCATCT TTGAGATAGGTTAAGAAATTGAATTGGCACGTAAA	2497
	AGTCCAATCCATGTTTA	2498
	TAAACATGGATTGGACT	2499
Haemophilia B Pro55Arg CCA-CGA	TTACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGA TCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAA GGATGACATTAATTCCTATGAATGTTGGTGTCCCTT	2500
	AAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAAC TGCCGCCATTTAAACATGGATTGGACTCACACTGATCTCCATC TTTGAGATAGGTTAAGAAATTGAATTGGCACGTAA	2501

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GTCCAATC**C**ATGTTTAA	2502
	TTAAACAT**G**GATTGGAC	2503
Haemophilia B Pro55Gln CCA-CAA	TTACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGA TCAGTGTGAGTCCAATC**C**ATGTTTAAATGGCGGCAGTTGCAA GGATGACATTAATTCCTATGAATGTTGGTGTCCCTT	2504
	AAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAAC TGCCGCCATTTAAACAT**G**GATTGGACTCACACTGATCTCCATC TTTGAGATAGGTTAAGAAATTGAATTGGCACGTAA	2505
	GTCCAATC**C**ATGTTTAA	2506
	TTAAACAT**G**GATTGGAC	2507
Haemophilia B Pro55Leu CCA-CTA	TTACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGA TCAGTGTGAGTCCAATC**C**ATGTTTAAATGGCGGCAGTTGCAA GGATGACATTAATTCCTATGAATGTTGGTGTCCCTT	2508
	AAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAAC TGCCGCCATTTAAACAT**G**GATTGGACTCACACTGATCTCCATC TTTGAGATAGGTTAAGAAATTGAATTGGCACGTAA	2509
	GTCCAATC**C**ATGTTTAA	2510
	TTAAACAT**G**GATTGGAC	2511
Haemophilia B Pro55Ser tCCA-TCA	TTTACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAG ATCAGTGTGAGTCCAAT**C**CATGTTTAAATGGCGGCAGTTGCA AGGATGACATTAATTCCTATGAATGTTGGTGTCCCT	2512
	AGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAACT GCCGCCATTTAAACATG**G**ATTGGACTCACACTGATCTCCATCT TTGAGATAGGTTAAGAAATTGAATTGGCACGTAAA	2513
	AGTCCAAT**C**CATGTTTA	2514
	TAAACATG**G**ATTGGACT	2515
Haemophilia B Cys56Arg aTGT-CGT	ACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGATC AGTGTGAGTCCAATCCA**T**GTTTAAATGGCGGCAGTTGCAAGG ATGACATTAATTCCTATGAATGTTGGTGTCCCTTTG	2516
	CAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCA ACTGCCGCCATTTAAAC**A**TGGATTGGACTCACACTGATCTCC ATCTTTGAGATAGGTTAAGAAATTGAATTGGCACGT	2517
	CCAATCCA**T**GTTTAAAT	2518
	ATTTAAAC**A**TGGATTGG	2519
Haemophilia B Cys56Ser aTGT-AGT	ACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGATC AGTGTGAGTCCAATCCA**T**GTTTAAATGGCGGCAGTTGCAAGG ATGACATTAATTCCTATGAATGTTGGTGTCCCTTTG	2520
	CAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCA ACTGCCGCCATTTAAAC**A**TGGATTGGACTCACACTGATCTCC ATCTTTGAGATAGGTTAAGAAATTGAATTGGCACGT	2521

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCAATCCATGTTTAAAT	2522
	ATTTAAACATGGATTGG	2523
Haemophilia B Cys56Ser TGT-TCT	CGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGATCA GTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGA TGACATTAATTCCTATGAATGTTGGTGTCCCTTTGG	2524
	CCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGC AACTGCCGCCATTTAAACATGGATTGGACTCACACTGATCTCC ATCTTTGAGATAGGTTAAGAAATTGAATTGGCACG	2525
	CAATCCATGTTTAAATG	2526
	CATTTAAACATGGATTG	2527
Haemophilia B Cys56Tyr TGT-TAT	CGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGATCA GTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGA TGACATTAATTCCTATGAATGTTGGTGTCCCTTTGG	2528
	CCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGC AACTGCCGCCATTTAAACATGGATTGGACTCACACTGATCTCC ATCTTTGAGATAGGTTAAGAAATTGAATTGGCACG	2529
	CAATCCATGTTTAAATG	2530
	CATTTAAACATGGATTG	2531
Haemophilia B Asn58Lys AATg-AAG	ATTCAATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAG TCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTA ATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAA	2532
	TTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTCA TCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCACACT GATCTCCATCTTTGAGATAGGTTAAGAAATTGAAT	2533
	TGTTTAAATGGCGGCAG	2534
	CTGCCGCCATTTAAACA	2535
Haemophilia B Gly59Asp GGC-GAC	TCAATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTC CAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAAT TCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGG	2536
	CCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGT CATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCACA CTGATCTCCATCTTTGAGATAGGTTAAGAAATTGA	2537
	TTTAAATGGCGGCAGTT	2538
	AACTGCCGCCATTTAAA	2539
Haemophilia B Gly59Val GGC-GTC	TCAATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTC CAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAAT TCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGG	2540
	CCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGT CATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCACA CTGATCTCCATCTTTGAGATAGGTTAAGAAATTGA	2541

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTTAAATGGCGGCAGTT	2542
	AACTGCCGCCATTTAAA	2543
Haemophilia B Gly59Ser tGGC-AGC	TTCAATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGT CCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAA TTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAG	2544
	CTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTC ATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCACAC TGATCTCCATCTTTGAGATAGGTTAAGAAATTGAA	2545
	GTTTAAATGGCGGCAGT	2546
	ACTGCCGCCATTTAAAC	2547
Haemophilia B Gly60Ser cGGC-AGC	AATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCA ATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTC CTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAA	2548
	TTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAAT GTCATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCA CACTGATCTCCATCTTTGAGATAGGTTAAGAAATT	2549
	TAAATGGCGGCAGTTGC	2550
	GCAACTGCCGCCATTTA	2551
Haemophilia B Gly60Cys cGGC-TGC	AATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCA ATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTC CTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAA	2552
	TTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAAT GTCATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCA CACTGATCTCCATCTTTGAGATAGGTTAAGAAATT	2553
	TAAATGGCGGCAGTTGC	2554
	GCAACTGCCGCCATTTA	2555
Haemophilia B Gly60Asp GGC-GAC	ATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCAA TCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTCC TATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAA	2556
	TTTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAA TGTCATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTC ACACTGATCTCCATCTTTGAGATAGGTTAAGAAAT	2557
	AAATGGCGGCAGTTGCA	2558
	TGCAACTGCCGCCATTT	2559
Haemophilia B Gly60Arg cGGC-CGC	AATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCA ATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTC CTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAA	2560
	TTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAAT GTCATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCA CACTGATCTCCATCTTTGAGATAGGTTAAGAAATT	2561

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAAATGGCGGCAGTTGC	2562
	GCAACTGCCGCCATTTA	2563
Haemophilia B Cys62Tyr TGC-TAC	TAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATG TTTAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAA TGTTGGTGTCCCTTTGGATTTGAAGGAAAGAACTG	2564
	CAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAGG AATTAATGTCATCCTTGCAACTGCCGCCATTTAAACATGGATT GGACTCACACTGATCTCCATCTTTGAGATAGGTTA	2565
	CGGCAGTTGCAAGGATG	2566
	CATCCTTGCAACTGCCG	2567
Haemophilia B Cys62Ser TGC-TCC	TAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATG TTTAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAA TGTTGGTGTCCCTTTGGATTTGAAGGAAAGAACTG	2568
	CAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAGG AATTAATGTCATCCTTGCAACTGCCGCCATTTAAACATGGATT GGACTCACACTGATCTCCATCTTTGAGATAGGTTA	2569
	CGGCAGTTGCAAGGATG	2570
	CATCCTTGCAACTGCCG	2571
Haemophilia B Cys62Term TGCa-TGA	AACCTATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGT TTAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAAT GTTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGT	2572
	ACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAG GAATTAATGTCATCCTTGCAACTGCCGCCATTTAAACATGGAT TGGACTCACACTGATCTCCATCTTTGAGATAGGTT	2573
	GGCAGTTGCAAGGATGA	2574
	TCATCCTTGCAACTGCC	2575
Haemophilia B Asp64Glu GATg-GAG	TCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAAT GGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGG TGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTA	2576
	TAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACAT TCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAAC ATGGATTGGACTCACACTGATCTCCATCTTTGAGA	2577
	TGCAAGGATGACATTAA	2578
	TTAATGTCATCCTTGCA	2579
Haemophilia B Asp64Gly GAT-GGT	ATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAA TGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTG GTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATT	2580
	AATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATT CATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAACA TGGATTGGACTCACACTGATCTCCATCTTTGAGAT	2581

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTGCAAGGATGACATTA	2582
	TAATGTCATCCTTGCAA	2583
Haemophilia B Asp64Asn gGAT-AAT	TATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAA ATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTG GTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAAT	2584
	ATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTC ATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAACAT GGATTGGACTCACACTGATCTCCATCTTTGAGATA	2585
	GTTGCAAGGATGACATT	2586
	AATGTCATCCTTGCAAC	2587
Haemophilia B Ile66Ser ATT-AGT	AAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGGCG GCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGGTGTCC CTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAA	2588
	TTACCTAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACC AACATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATT TAAACATGGATTGGACTCACACTGATCTCCATCTT	2589
	GGATGACATTAATTCCT	2590
	AGGAATTAATGTCATCC	2591
Haemophilia B Ile66Thr ATT-ACT	AAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGGCG GCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGGTGTCC CTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAA	2592
	TTACCTAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACC AACATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATT TAAACATGGATTGGACTCACACTGATCTCCATCTT	2593
	GGATGACATTAATTCCT	2594
	AGGAATTAATGTCATCC	2595
Haemophilia B Asn67Lys AATt-AAA	TGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAG TTGCAAGGATGACATTAATTCCTATGAATGTTGGTGTCCCTTT GGATTTGAAGGAAAGAACTGTGAATTAGGTAAGTAA	2596
	TTACTTACCTAATTCACAGTTCTTTCCTTCAAATCCAAAGGGAC ACCAACATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCC ATTTAAACATGGATTGGACTCACACTGATCTCCA	2597
	GACATTAATTCCTATGA	2598
	TCATAGGAATTAATGTC	2599
Haemophilia B Tyr69Cys TAT-TGT	ATCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCA AGGATGACATTAATTCCTATGAATGTTGGTGTCCCTTTGGATT TGAAGGAAAGAACTGTGAATTAGGTAAGTAACTATT	2600
	AATAGTTACTTACCTAATTCACAGTTCTTTCCTTCAAATCCAAA GGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAACTG CCGCCATTTAAACATGGATTGGACTCACACTGAT	2601

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAATTCCTATGAATGTT	2602
	AACATTCATAGGAATTA	2603
Haemophilia B Cys71Term TGTt-TGA	TGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGA CATTAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGA AAGAACTGTGAATTAGGTAAGTAACTATTTTTTGAA	2604
	TTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTTCAA ATCCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTT GCAACTGCCGCCATTTAAACATGGATTGGACTCA	2605
	TATGAATGTTGGTGTCC	2606
	GGACACCAACATTCATA	2607
Haemophilia B Cys71Ser TGT-TCT	GTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATG ACATTAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGG AAAGAACTGTGAATTAGGTAAGTAACTATTTTTTGA	2608
	TCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTTCAAA TCCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTG CAACTGCCGCCATTTAAACATGGATTGGACTCAC	2609
	CTATGAATGTTGGTGTC	2610
	GACACCAACATTCATAG	2611
Haemophilia B Cys71Tyr TGT-TAT	GTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATG ACATTAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGG AAAGAACTGTGAATTAGGTAAGTAACTATTTTTTGA	2612
	TCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTTCAAA TCCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTG CAACTGCCGCCATTTAAACATGGATTGGACTCAC	2613
	CTATGAATGTTGGTGTC	2614
	GACACCAACATTCATAG	2615
Haemophilia B Cys71Ser aTGT-AGT	TGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGAT GACATTAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAG GAAAGAACTGTGAATTAGGTAAGTAACTATTTTTTG	2616
	CAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTTCAAAT CCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGC AACTGCCGCCATTTAAACATGGATTGGACTCACA	2617
	CCTATGAATGTTGGTGT	2618
	ACACCAACATTCATAGG	2619
Haemophilia B Trp72Arg tTGG-AGG	GAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGAC ATTAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAA AGAACTGTGAATTAGGTAAGTAACTATTTTTTGAAT	2620
	ATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTTCA AATCCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCT TGCAACTGCCGCCATTTAAACATGGATTGGACTC	2621

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATGAATGTTGGTGTCCC	2622
	GGGACACCAACATTCAT	2623
Haemophilia B Trp72Term TGGt-TGA	GTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACAT TAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAAG AACTGTGAATTAGGTAAGTAACTATTTTTTGAATAC	2624
	GTATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTT CAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTCATC CTTGCAACTGCCGCCATTTAAACATGGATTGGAC	2625
	GAATGTTGGTGTCCCTT	2626
	AAGGGACACCAACATTC	2627
Haemophilia B Cys73Tyr TGT-TAT	CCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAA TTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAAC TGTGAATTAGGTAAGTAACTATTTTTTGAATACTC	2628
	GAGTATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCC TTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTCA TCCTTGCAACTGCCGCCATTTAAACATGGATTGG	2629
	ATGTTGGTGTCCCTTTG	2630
	CAAAGGGACACCAACAT	2631
Haemophilia B Cys73Arg gTGT-CGT	TCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTA ATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAA CTGTGAATTAGGTAAGTAACTATTTTTTGAATACT	2632
	AGTATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCT TCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTCAT CCTTGCAACTGCCGCCATTTAAACATGGATTGGA	2633
	AATGTTGGTGTCCCTTT	2634
	AAAGGGACACCAACATT	2635
Haemophilia B Cys73Phe TGT-TTT	CCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAA TTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAAC TGTGAATTAGGTAAGTAACTATTTTTTGAATACTC	2636
	GAGTATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCC TTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTCA TCCTTGCAACTGCCGCCATTTAAACATGGATTGG	2637
	ATGTTGGTGTCCCTTTG	2638
	CAAAGGGACACCAACAT	2639
Haemophilia B Cys73Term TGTc-TGA	CAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAAT TCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAACT GTGAATTAGGTAAGTAACTATTTTTTGAATACTCA	2640
	TGAGTATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTC CTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTC ATCCTTGCAACTGCCGCCATTTAAACATGGATTG	2641
	TGTTGGTGTCCCTTTGG	2642

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCAAAGGGACACCAACA	2643
Haemophilia B Gly76Val GGA-GTA	GTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGA ATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTA GGTAAGTAACTATTTTTTGAATACTCATGGTTCAA	2644
	TTGAACCATGAGTATTCAAAAAATAGTTACTTACCTAATTCACA GTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAA TTAATGTCATCCTTGCAACTGCCGCCATTTAAAC	2645
	TCCCTTTGGATTTGAAG	2646
	CTTCAAATCCAAAGGGA	2647
Haemophilia B Gly76Arg tGGA-AGA	TGTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATG AATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATT AGGTAAGTAACTATTTTTTGAATACTCATGGTTCA	2648
	TGAACCATGAGTATTCAAAAAATAGTTACTTACCTAATTCACAG TTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAAT TAATGTCATCCTTGCAACTGCCGCCATTTAAACA	2649
	GTCCCTTTGGATTTGAA	2650
	TTCAAATCCAAAGGGAC	2651
Haemophilia B Phe77Cys TTT-TGT	TAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATG TTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGT AAGTAACTATTTTTTGAATACTCATGGTTCAAAGT	2652
	ACTTTGAACCATGAGTATTCAAAAAATAGTTACTTACCTAATTC ACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAG GAATTAATGTCATCCTTGCAACTGCCGCCATTTA	2653
	CTTTGGATTTGAAGGAA	2654
	TTCCTTCAAATCCAAAG	2655
Haemophilia B Phe77Ser TTT-TCT	TAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATG TTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGT AAGTAACTATTTTTTGAATACTCATGGTTCAAAGT	2656
	ACTTTGAACCATGAGTATTCAAAAAATAGTTACTTACCTAATTC ACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAG GAATTAATGTCATCCTTGCAACTGCCGCCATTTA	2657
	CTTTGGATTTGAAGGAA	2658
	TTCCTTCAAATCCAAAG	2659
Haemophilia B Phe77Tyr TTT-TAT	TAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATG TTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGT AAGTAACTATTTTTTGAATACTCATGGTTCAAAGT	2660
	ACTTTGAACCATGAGTATTCAAAAAATAGTTACTTACCTAATTC ACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAG GAATTAATGTCATCCTTGCAACTGCCGCCATTTA	2661
	CTTTGGATTTGAAGGAA	2662

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTCCTTCAAATCCAAAG	2663
Haemophilia B Glu78Lys tGAA-AAA	AATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTT GGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAA GTAACTATTTTTTGAATACTCATGGTTCAAAGTTT	2664
	AAACTTTGAACCATGAGTATTCAAAAAATAGTTACTTACCTAAT TCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCAT AGGAATTAATGTCATCCTTGCAACTGCCGCCATT	2665
	TTGGATTTGAAGGAAAG	2666
	CTTTCCTTCAAATCCAA	2667
Haemophilia B Gly79Val GGA-GTA	GCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGGT GTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAAGTA ACTATTTTTTGAATACTCATGGTTCAAAGTTTCCCT	2668
	AGGGAAACTTTGAACCATGAGTATTCAAAAAATAGTTACTTAC CTAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACA TTCATAGGAATTAATGTCATCCTTGCAACTGCCGC	2669
	ATTTGAAGGAAAGAACT	2670
	AGTTCTTTCCTTCAAAT	2671
Haemophilia B Gly79Arg aGGA-AGA	GGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGG TGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAAGT AACTATTTTTTGAATACTCATGGTTCAAAGTTTCCC	2672
	GGGAAACTTTGAACCATGAGTATTCAAAAAATAGTTACTTACC TAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACAT TCATAGGAATTAATGTCATCCTTGCAACTGCCGCC	2673
	GATTTGAAGGAAAGAAC	2674
	GTTCTTTCCTTCAAATC	2675
Haemophilia B Gly79Glu GGA-GAA	GCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGGT GTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAAGTA ACTATTTTTTGAATACTCATGGTTCAAAGTTTCCCT	2676
	AGGGAAACTTTGAACCATGAGTATTCAAAAAATAGTTACTTAC CTAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACA TTCATAGGAATTAATGTCATCCTTGCAACTGCCGC	2677
	ATTTGAAGGAAAGAACT	2678
	AGTTCTTTCCTTCAAAT	2679
Haemophilia B Cys88Ser TGT-TCT	TTAGAAATGCATGTTAAATGATGCTGTTACTGTCTATTTTGCTT CTTTTAGATGTAACATGTAACATTAAGAATGGCAGATGCGAGC AGTTTTGTAAAAATAGTGCTGATAACAAGGTGGT	2680
	ACCACCTTGTTATCAGCACTATTTTTACAAAACTGCTCGCATC TGCCATTCTTAATGTTACATGTTACATCTAAAAGAAGCAAAATA GACAGTAACAGCATCATTTAACATGCATTTCTAA	2681
	TGTAACATGTAACATTA	2682

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAATGTTACATGTTACA	2683
Haemophilia B Cys88Phe TGT-TTT	TTAGAAATGCATGTTAAATGATGCTGTTACTGTCTATTTTGCTT CTTTTAGATGTAACATGTAACATTAAGAATGGCAGATGCGAGC AGTTTTGTAAAAATAGTGCTGATAACAAGGTGGT	2684
	ACCACCTTGTTATCAGCACTATTTTTACAAAACTGCTCGCATC TGCCATTCTTAATGTTACATGTTACATCTAAAAGAAGCAAAATA GACAGTAACAGCATCATTTAACATGCATTTCTAA	2685
	TGTAACATGTAACATTA	2686
	TAATGTTACATGTTACA	2687
Haemophilia B Cys88Arg aTGT-CGT	TTTAGAAATGCATGTTAAATGATGCTGTTACTGTCTATTTTGCT TCTTTTAGATGTAACATGTAACATTAAGAATGGCAGATGCGAG CAGTTTTGTAAAAATAGTGCTGATAACAAGGTGG	2688
	CCACCTTGTTATCAGCACTATTTTTACAAAACTGCTCGCATCT GCCATTCTTAATGTTACATGTTACATCTAAAAGAAGCAAAATA GACAGTAACAGCATCATTTAACATGCATTTCTAAA	2689
	ATGTAACATTGTAACATT	2690
	AATGTTACATGTTACAT	2691
Haemophilia B Cys88Tyr TGT-TAT	TTAGAAATGCATGTTAAATGATGCTGTTACTGTCTATTTTGCTT CTTTTAGATGTAACATGTAACATTAAGAATGGCAGATGCGAGC AGTTTTGTAAAAATAGTGCTGATAACAAGGTGGT	2692
	ACCACCTTGTTATCAGCACTATTTTTACAAAACTGCTCGCATC TGCCATTCTTAATGTTACATGTTACATCTAAAAGAAGCAAAATA GACAGTAACAGCATCATTTAACATGCATTTCTAA	2693
	TGTAACATGTAACATTA	2694
	TAATGTTACATGTTACA	2695
Haemophilia B Ile90Thr ATT-ACT	ATGCATGTTAAATGATGCTGTTACTGTCTATTTTGCTTCTTTTA GATGTAACATGTAACATTAAGAATGGCAGATGCGAGCAGTTTT GTAAAAATAGTGCTGATAACAAGGTGGTTTGCTC	2696
	GAGCAAACCACCTTGTTATCAGCACTATTTTTACAAAACTGCT CGCATCTGCCATTCTTAATGTTACATGTTACATCTAAAAGAAG CAAAATAGACAGTAACAGCATCATTTAACATGCAT	2697
	ATGTAACATTAAGAATG	2698
	CATTCTTAATGTTACAT	2699
Haemophilia B Asn92His gAAT-CAT	TGTTAAATGATGCTGTTACTGTCTATTTTGCTTCTTTTAGATGT AACATGTAACATTAAGAATGGCAGATGCGAGCAGTTTTGTAAA AATAGTGCTGATAACAAGGTGGTTTGCTCCTGTA	2700
	TACAGGAGCAAACCACCTTGTTATCAGCACTATTTTTACAAAA CTGCTCGCATCTGCCATTCTTAATGTTACATGTTACATCTAAAA GAAGCAAAATAGACAGTAACAGCATCATTTAACA	2701
	ACATTAAGAATGGCAGA	2702

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCTGCCATTCTTAATGT	2703
Haemophilia B Asn92Lys AATg-AAA	TTAAATGATGCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAA CATGTAACATTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAA TAGTGCTGATAACAAGGTGGTTTGCTCCTGTACT	2704
	AGTACAGGAGCAAACCACCTTGTTATCAGCACTATTTTTACAA AACTGCTCGCATCTGCCATTCTTAATGTTACATGTTACATCTA AAAGAAGCAAAATAGACAGTAACAGCATCATTTAA	2705
	ATTAAGAATGGCAGATG	2706
	CATCTGCCATTCTTAAT	2707
Haemophilia B Gly93Asp GGC-GAC	AAATGATGCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAACA TGTAACATTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAATA GTGCTGATAACAAGGTGGTTTGCTCCTGTACTGA	2708
	TCAGTACAGGAGCAAACCACCTTGTTATCAGCACTATTTTTAC AAAACTGCTCGCATCTGCCATTCTTAATGTTACATGTTACATCT AAAAGAAGCAAAATAGACAGTAACAGCATCATTT	2709
	TAAGAATGGCAGATGCG	2710
	CGCATCTGCCATTCTTA	2711
Haemophilia B Gly93Ser tGGC-AGC	TAAATGATGCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAAC ATGTAACATTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAAT AGTGCTGATAACAAGGTGGTTTGCTCCTGTACTG	2712
	CAGTACAGGAGCAAACCACCTTGTTATCAGCACTATTTTTACA AAACTGCTCGCATCTGCCATTCTTAATGTTACATGTTACATCTA AAAGAAGCAAAATAGACAGTAACAGCATCATTTA	2713
	TTAAGAATGGCAGATGC	2714
	GCATCTGCCATTCTTAA	2715
Haemophilia B Arg94Ser AGAt-AGT	GATGCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAACATGTA ACATTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGC TGATAACAAGGTGGTTTGCTCCTGTACTGAGGGA	2716
	TCCCTCAGTACAGGAGCAAACCACCTTGTTATCAGCACTATTT TTACAAAACTGCTCGCATCTGCCATTCTTAATGTTACATGTTAC ATCTAAAAGAAGCAAAATAGACAGTAACAGCATC	2717
	AATGGCAGATGCGAGCA	2718
	TGCTCGCATCTGCCATT	2719
Haemophilia B Cys95Tyr TGC-TAC	TGCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAACATGTAAC ATTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGCTG ATAACAAGGTGGTTTGCTCCTGTACTGAGGGATA	2720
	TATCCCTCAGTACAGGAGCAAACCACCTTGTTATCAGCACTAT TTTTACAAAACTGCTCGCATCTGCCATTCTTAATGTTACATGTT ACATCTAAAAGAAGCAAAATAGACAGTAACAGCA	2721
	TGGCAGATGCGAGCAGT	2722

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACTGCTCGCATCTGCCA	2723
Haemophilia B Cys95Trp TGCg-TGG	GCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAACATGTAACA TTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGCTGA TAACAAGGTGGTTTGCTCCTGTACTGAGGGATAT	2724
	ATATCCCTCAGTACAGGAGCAAACCACCTTGTTATCAGCACTA TTTTTACAAAACTGCTCGCATCTGCCATTCTTAATGTTACATGT TACATCTAAAAGAAGCAAAATAGACAGTAACAGC	2725
	GGCAGATGCGAGCAGTT	2726
	AACTGCTCGCATCTGCC	2727
Haemophilia B Cys95Term TGCg-TGA	GCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAACATGTAACA TTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGCTGA TAACAAGGTGGTTTGCTCCTGTACTGAGGGATAT	2728
	ATATCCCTCAGTACAGGAGCAAACCACCTTGTTATCAGCACTA TTTTTACAAAACTGCTCGCATCTGCCATTCTTAATGTTACATGT TACATCTAAAAGAAGCAAAATAGACAGTAACAGC	2729
	GGCAGATGCGAGCAGTT	2730
	AACTGCTCGCATCTGCC	2731
Haemophilia B Gln97Pro CAG-CCG	TACTGTCTATTTTGCTTCTTTTAGATGTAACATGTAACATTAAG AATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGCTGATAACA AGGTGGTTTGCTCCTGTACTGAGGGATATCGACT	2732
	AGTCGATATCCCTCAGTACAGGAGCAAACCACCTTGTTATCA GCACTATTTTTACAAAACTGCTCGCATCTGCCATTCTTAATGTT ACATGTTACATCTAAAAGAAGCAAAATAGACAGTA	2733
	ATGCGAGCAGTTTTGTA	2734
	TACAAAACTGCTCGCAT	2735
Haemophilia B Gln97Glu gCAG-GAG	TTACTGTCTATTTTGCTTCTTTTAGATGTAACATGTAACATTAA GAATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGCTGATAAC AAGGTGGTTTGCTCCTGTACTGAGGGATATCGAC	2736
	GTCGATATCCCTCAGTACAGGAGCAAACCACCTTGTTATCAG CACTATTTTTACAAAACTGCTCGCATCTGCCATTCTTAATGTTA CATGTTACATCTAAAAGAAGCAAAATAGACAGTAA	2737
	GATGCGAGCAGTTTTGT	2738
	ACAAAACTGCTCGCATC	2739
Haemophilia B Cys99Arg tTGT-CGT	TCTATTTTGCTTCTTTTAGATGTAACATGTAACATTAAGAATGG CAGATGCGAGCAGTTTTGTAAAAATAGTGCTGATAACAAGGTG GTTTGCTCCTGTACTGAGGGATATCGACTTGCAG	2740
	CTGCAAGTCGATATCCCTCAGTACAGGAGCAAACCACCTTGT TATCAGCACTATTTTTACAAAACTGCTCGCATCTGCCATTCTT AATGTTACATGTTACATCTAAAAGAAGCAAAATAGA	2741
	AGCAGTTTTGTAAAAAT	2742

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATTTTTACAAAACTGCT	2743
Haemophilia B Cys99Tyr TGT-TAT	CTATTTTGCTTCTTTTAGATGTAACATGTAACATTAAGAATGGC AGATGCGAGCAGTTTTGTAAAAATAGTGCTGATAACAAGGTG GTTTGCTCCTGTACTGAGGGATATCGACTTGCAGA	2744
	TCTGCAAGTCGATATCCCTCAGTACAGGAGCAAACCACCTTG TTATCAGCACTATTTTTACAAAACTGCTCGCATCTGCCATTCTT AATGTTACATGTTACATCTAAAAGAAGCAAAATAG	2745
	GCAGTTTTGTAAAAATA	2746
	TATTTTTACAAAACTGC	2747
Warfarin sensitivity Ala(-10)Thr cGCC-ACC	TTTTTTGCTAAAACTAAAGAATTATTCTTTTACATTTCAGTTTTT CTTGATCATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGA GGTATAATTCAGGTAAATTGGAAGAGTTTGTTC	2748
	GAACAAACTCTTCCAATTTACCTGAATTATACCTCTTTGGCCG ATTCAGAATTTTGTTGGCGTTTTCATGATCAAGAAAAACTGAAA TGTAAAAGAATAATTCTTTAGTTTTAGCAAAAAA	2749
	ATGAAAACGCCAACAAA	2750
	TTTGTTGGCGTTTTCAT	2751
Warfarin sensitivity Ala(-10)Val GCC-GTC	TTTTTGCTAAAACTAAAGAATTATTCTTTTACATTTCAGTTTTTC TTGATCATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAG GTATAATTCAGGTAAATTGGAAGAGTTTGTTCA	2752
	TGAACAAACTCTTCCAATTTACCTGAATTATACCTCTTTGGCC GATTCAGAATTTTGTTGGCGTTTTCATGATCAAGAAAAACTGA AATGTAAAAGAATAATTCTTTAGTTTTAGCAAAAA	2753
	TGAAAACGCCAACAAAA	2754
	TTTTGTTGGCGTTTTCA	2755
Haemophilia B Gly(-26)Val GGA-GTA	TGCAGCGCGTGAACATGATCATGGCAGAATCACCAGGCCTCA TCACCATCTGCCTTTTAGGATATCTACTCAGTGCTGAATGTAC AGGTTTGTTTCCTTTTTTAAAATACATTGAGTATGC	2756
	GCATACTCAATGTATTTTAAAAAAGGAAACAAACCTGTACATTC AGCACTGAGTAGATATCCTAAAAGGCAGATGGTGATGAGGCC TGGTGATTCTGCCATGATCATGTTCACGCGCTGCA	2757
	CCTTTTAGGATATCTAC	2758
	GTAGATATCCTAAAAGG	2759
Haemophilia B Leu(-27)Term TTA-TAA	TTATGCAGCGCGTGAACATGATCATGGCAGAATCACCAGGCC TCATCACCATCTGCCTTTTAGGATATCTACTCAGTGCTGAATG TACAGGTTTGTTTCCTTTTTTAAAATACATTGAGTA	2760
	TACTCAATGTATTTTAAAAAAGGAAACAAACCTGTACATTCAGC ACTGAGTAGATATCCTAAAAGGCAGATGGTGATGAGGCCTGG TGATTCTGCCATGATCATGTTCACGCGCTGCATAA	2761
	CTGCCTTTTAGGATATC	2762

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GATATCCTAAAAGGCAG	2763
Haemophilia B Ile(-30)Asn ATC-AAC	TAGCAAAGGTTATGCAGCGCGTGAACATGATCATGGCAGAAT CACCAGGCCTCATCACCATCTGCCTTTTAGGATATCTACTCAG TGCTGAATGTACAGGTTTGTTTCCTTTTTTAAAATA	2764
	TATTTTAAAAAAGGAAACAAACCTGTACATTCAGCACTGAGTA GATATCCTAAAAGGCAGATGGTGATGAGGCCTGGTGATTCTG CCATGATCATGTTCACGCGCTGCATAACCTTTGCTA	2765
	CATCACCATCTGCCTTT	2766
	AAAGGCAGATGGTGATG	2767
Haemophilia B Ile(-40)Phe gATC-TTC	ACTAATCGACCTTACCACTTTCACAATCTGCTAGCAAAGGTTA TGCAGCGCGTGAACATGATCATGGCAGAATCACCAGGCCTCA TCACCATCTGCCTTTTAGGATATCTACTCAGTGCTG	2768
	CAGCACTGAGTAGATATCCTAAAAGGCAGATGGTGATGAGGC CTGGTGATTCTGCCATGATCATGTTCACGCGCTGCATAACCTT TGCTAGCAGATTGTGAAAGTGGTAAGGTCGATTAGT	2769
	TGAACATGATCATGGCA	2770
	TGCCATGATCATGTTCA	2771
Haemophilia B Arg(-44)His CGC-CAC	ACTTTGGTACAACTAATCGACCTTACCACTTTCACAATCTGCT AGCAAAGGTTATGCAGCGCGTGAACATGATCATGGCAGAATC ACCAGGCCTCATCACCATCTGCCTTTTAGGATATCT	2772
	AGATATCCTAAAAGGCAGATGGTGATGAGGCCTGGTGATTCT GCCATGATCATGTTCACGCGCTGCATAACCTTTGCTAGCAGA TTGTGAAAGTGGTAAGGTCGATTAGTTGTACCAAAGT	2773
	TATGCAGCGCGTGAACA	2774
	TGTTCACGCGCTGCATA	2775

EXAMPLE 15
Alpha thalassemia - Hemoglobin alpha locus 1

The thalassemia syndromes are a heterogeneous group of inherited anemias characterized by defects in the synthesis of one or more globin chain subunits. For example, beta-thalassemia discussed in Example 6, is caused by a decrease in beta-chain production relative to alpha-chain production; the converse is the case for alpha-thalassemia. The attached table discloses the correcting oligonucleotide base sequences for the hemoglobin alpha locus 1 oligonucleotides of the invention.

Table 22
HBA1 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia alpha Met(-1)Val cATG-GTG	CCCTGGCGCGCTCGCGGCCCGGCACTCTTCTGGTCCCCACA GACTCAGAGAGAACCCACCATGGTGCTGTCTCCTGCCGACA AGACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGC	2776
	GCGCGCCGACCTTACCCCAGGCGGCCTTGACGTTGGTCTTG TCGGCAGGAGACAGCACCATGGTGGGTTCTCTCTGAGTCTGT GGGGACCAGAAGAGTGCCGGGCCGCGAGCGCGCCAGGG	2777
	AACCCACCATGGTGCTG	2778
	CAGCACCATGGTGGGTT	2779
Haemoglobin variant Ala12Asp GCC-GAC	CACAGACTCAGAGAGAACCCACCATGGTGCTGTCTCCTGCC GACAAGACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGC GCACGCTGGCGAGTATGGTGCGGAGGCCCTGGAGAGGTG	2780
	CACCTCTCCAGGGCCTCCGCACCATACTCGCCAGCGTGCGC GCCGACCTTACCCCAGGCGGCCTTGACGTTGGTCTTGTCGG CAGGAGACAGCACCATGGTGGGTTCTCTCTGAGTCTGTG	2781
	CGTCAAGGCCGCCTGGG	2782
	CCCAGGCGGCCTTGACG	2783
Haemoglobin variant Gly15Asp GGT-GAT	AGAGAGAACCCACCATGGTGCTGTCTCCTGCCGACAAGACCA ACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCACGCTGG CGAGTATGGTGCGGAGGCCCTGGAGAGGTGAGGCTCCCT	2784
	AGGGAGCCTCACCTCTCCAGGGCCTCCGCACCATACTCGCC AGCGTGCGCGCCGACCTTACCCCAGGCGGCCTTGACGTTGG TCTTGTCGGCAGGAGACAGCACCATGGTGGGTTCTCTCT	2785
	CGCCTGGGGTAAGGTCG	2786
	CGACCTTACCCCAGGCG	2787
Haemoglobin variant Tyr24Cys TAT-TGT	CTGCCGACAAGACCAACGTCAAGGCCGCCTGGGGTAAGGTC GGCGCGCACGCTGGCGAGTATGGTGCGGAGGCCCTGGAGA GGTGAGGCTCCCTCCCCTGCTCCGACCCGGGCTCCTCGCC	2788
	GGCGAGGAGCCCGGGTCGGAGCAGGGGAGGGAGCCTCACC TCTCCAGGGCCTCCGCACCATACTCGCCAGCGTGCGCGCCG ACCTTACCCCAGGCGGCCTTGACGTTGGTCTTGTCGGCAG	2789
	TGGCGAGTATGGTGCGG	2790
	CCGCACCATACTCGCCA	2791
Haemoglobin variant Glu27Asp GAGg-GAT	GACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCAC GCTGGCGAGTATGGTGCGGAGGCCCTGGAGAGGTGAGGCT CCCTCCCCTGCTCCGACCCGGGCTCCTCGCCCGCCCGGAC C	2792
	GGTCCGGGCGGGCGAGGAGCCCGGGTCGGAGCAGGGGAG GGAGCCTCACCTCTCCAGGGCCTCCGCACCATACTCGCCAG CGTGCGCGCCGACCTTACCCCAGGCGGCCTTGACGTTGGTC	2793
	GGTGCGGAGGCCCTGGA	2794
	TCCAGGGCCTCCGCACC	2795

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemoglobin variant Asn68Lys AACg-AAG	GAGCCACGGCTCTGCCCAGGTTAAGGGCCACGGCAAGAAGG TGGCCGACGCGCTGACCAA**C**GCCGTGGCGCACGTGGACGA CATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCACGCG	2796
	CGCGTGCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATG TCGTCCACGTGCGCCACGGC**G**TTGGTCAGCGCGTCGGCCAC CTTCTTGCCGTGGCCCTTAACCTGGGCAGAGCCGTGGCTC	2797
	CTGACCAA**C**GCCGTGGC	2798
	GCCACGGC**G**TTGGTCAG	2799
Haemoglobin variant Asp74Gly GAC-GGC	AGGTTAAGGGCCACGGCAAGAAGGTGGCCGACGCGCTGACC AACGCCGTGGCGCACGTGG**A**CGACATGCCCAACGCGCTGTC CGCCCTGAGCGACCTGCACGCGCACAAGCTTCGGGTGGA	2800
	TCCACCCGAAGCTTGTGCGCGTGCAGGTCGCTCAGGGCGGA CAGCGCGTTGGGCATGTCG**T**CCACGTGCGCCACGGCGTTGG TCAGCGCGTCGGCCACCTTCTTGCCGTGGCCCTTAACCT	2801
	GCACGTGG**A**CGACATGC	2802
	GCATGTCG**T**CCACGTGC	2803
Haemoglobin variant Asp74His gGAC-CAC	CAGGTTAAGGGCCACGGCAAGAAGGTGGCCGACGCGCTGAC CAACGCCGTGGCGCACGTG**G**ACGACATGCCCAACGCGCTGT CCGCCCTGAGCGACCTGCACGCGCACAAGCTTCGGGTGG	2804
	CCACCCGAAGCTTGTGCGCGTGCAGGTCGCTCAGGGCGGAC AGCGCGTTGGGCATGTCGT**C**CACGTGCGCCACGGCGTTGGT CAGCGCGTCGGCCACCTTCTTGCCGTGGCCCTTAACCTG	2805
	CGCACGTG**G**ACGACATG	2806
	CATGTCGT**C**CACGTGCG	2807
Haemoglobin variant Asn78His cAAC-CAC	CACGGCAAGAAGGTGGCCGACGCGCTGACCAACGCCGTGG CGCACGTGGACGACATGCCC**A**ACGCGCTGTCCGCCCTGAGC GACCTGCACGCGCACAAGCTTCGGGTGGACCCGGTCAACT	2808
	AGTTGACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGTCG CTCAGGGCGGACAGCGCGT**T**GGGCATGTCGTCCACGTGCGC CACGGCGTTGGTCAGCGCGTCGGCCACCTTCTTGCCGTG	2809
	ACATGCCC**A**ACGCGCTG	2810
	CAGCGCGT**T**GGGCATGT	2811
Haemoglobin variant His87Tyr gCAC-TAC	ACCAACGCCGTGGCGCACGTGGACGACATGCCCAACGCGCT GTCCGCCCTGAGCGACCTG**C**ACGCGCACAAGCTTCGGGTGG ACCCGGTCAACTTCAAGGTGAGCGGCGGGCCGGGAGCGA	2812
	TCGCTCCCGGCCCGCCGCTCACCTTGAAGTTGACCGGGTCC ACCCGAAGCTTGTGCGCGT**G**CAGGTCGCTCAGGGCGGACAG CGCGTTGGGCATGTCGTCCACGTGCGCCACGGCGTTGGT	2813
	GCGACCTG**C**ACGCGCAC	2814
	GTGCGCGT**G**CAGGTCGC	2815
Haemoglobin variant Lys90Asn AAGc-AAC	GGCGCACGTGGACGACATGCCCAACGCGCTGTCCGCCCTGA GCGACCTGCACGCGCACAA**G**CTTCGGGTGGACCCGGTCAAC TTCAAGGTGAGCGGCGGGCCGGGAGCGATCTGGGTCGAG	2816

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTCGACCCAGATCGCTCCCGGCCCGCCGCTCACCTTGAAGT TGACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGTCGCTC AGGGCGGACAGCGCGTTGGGCATGTCGTCCACGTGCGCC	2817
	GCGCACAAGCTTCGGGT	2818
	ACCCGAAGCTTGTGCGC	2819
Haemoglobin variant Lys90Thr AAG-ACG	TGGCGCACGTGGACGACATGCCCAACGCGCTGTCCGCCCTG AGCGACCTGCACGCGCACAAGCTTCGGGTGGACCCGGTCAA CTTCAAGGTGAGCGGCGGGCCGGGAGCGATCTGGGTCGA	2820
	TCGACCCAGATCGCTCCCGGCCCGCCGCTCACCTTGAAGTT GACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGTCGCTCA GGGCGGACAGCGCGTTGGGCATGTCGTCCACGTGCGCCA	2821
	CGCGCACAAGCTTCGGG	2822
	CCCGAAGCTTGTGCGCG	2823
Haemoglobin variant Arg92Gln CGG-CAG	ACGTGGACGACATGCCCAACGCGCTGTCCGCCCTGAGCGAC CTGCACGCGCACAAGCTTCGGGTGGACCCGGTCAACTTCAA GGTGAGCGGCGGGCCGGGAGCGATCTGGGTCGAGGGGCG	2824
	CGCCCCTCGACCCAGATCGCTCCCGGCCCGCCGCTCACCTT GAAGTTGACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGT CGCTCAGGGCGGACAGCGCGTTGGGCATGTCGTCCACGT	2825
	CAAGCTTCGGGTGGACC	2826
	GGTCCACCCGAAGCTTG	2827
Haemoglobin variant Asp94Gly GAC-GGC	ACGACATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCAC GCGCACAAGCTTCGGGTGGACCCGGTCAACTTCAAGGTGAG CGGCGGGCCGGGAGCGATCTGGGTCGAGGGGCGAGATGG	2828
	CCATCTCGCCCCTCGACCCAGATCGCTCCCGGCCCGCCGCT CACCTTGAAGTTGACCGGGTCCACCCGAAGCTTGTGCGCGT GCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGTCGT	2829
	TCGGGTGGACCCGGTCA	2830
	TGACCGGGTCCACCCGA	2831
Haemoglobin variant Pro95Arg CCG-CGG	ACATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCACGCG CACAAGCTTCGGGTGGACCCGGTCAACTTCAAGGTGAGCGG CGGGCCGGGAGCGATCTGGGTCGAGGGGCGAGATGGCGC	2832
	GCGCCATCTCGCCCCTCGACCCAGATCGCTCCCGGCCCGCC GCTCACCTTGAAGTTGACCGGGTCCACCCGAAGCTTGTGCG CGTGCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGT	2833
	GGTGGACCCGGTCAACT	2834
	AGTTGACCGGGTCCACC	2835
Haemoglobin variant Ser102Arg AGCc-AGA	CGGCGGCTGCGGGCCTGGGCCCTCGGCCCCACTGACCCTC TTCTCTGCACAGCTCCTAAGCCACTGCCTGCTGGTGACCCTG GCCGCCCACCTCCCCGCCGAGTTCACCCCTGCGGTGCAC	2836
	GTGCACCGCAGGGGTGAACTCGGCGGGGAGGTGGGCGGCC AGGGTCACCAGCAGGCAGTGGCTTAGGAGCTGTGCAGAGAA GAGGGTCAGTGGGGCCGAGGGCCCAGGCCCGCAGCCGCCG	2837
	CTCCTAAGCCACTGCCT	2838

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGGCAGTGGCTTAGGAG	2839
Haemoglobin variant Glu116Lys cGAG-AAG	TTCTCTGCACAGCTCCTAAGCCACTGCCTGCTGGTGACCCTG GCCGCCCACCTCCCCGCCGAGTTCACCCCTGCGGTGCACGC CTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTGC	2840
	GCACGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCG TGCACCGCAGGGGTGAACTCGGCGGGGAGGTGGGCGGCCA GGGTCACCAGCAGGCAGTGGCTTAGGAGCTGTGCAGAGAA	2841
	TCCCCGCCGAGTTCACC	2842
	GGTGAACTCGGCGGGGA	2843
Haemoglobin variant Ala120Glu GCG-GAG	TCCTAAGCCACTGCCTGCTGGTGACCCTGGCCGCCCACCTC CCCGCCGAGTTCACCCCTGCGGTGCACGCCTCCCTGGACAA GTTCCTGGCTTCTGTGAGCACCGTGCTGACCTCCAAATA	2844
	TATTTGGAGGTCAGCACGGTGCTCACAGAAGCCAGGAACTTG TCCAGGGAGGCGTGCACCGCAGGGGTGAACTCGGCGGGGA GGTGGGCGGCCAGGGTCACCAGCAGGCAGTGGCTTAGGA	2845
	CACCCCTGCGGTGCACG	2846
	CGTGCACCGCAGGGGTG	2847
Thalassaemia alpha Leu129Pro CTG-CCG	TGGCCGCCCACCTCCCCGCCGAGTTCACCCCTGCGGTGCAC GCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTGCTG ACCTCCAAATACCGTTAAGCTGGAGCCTCGGTGGCCAT	2848
	ATGGCCACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAGC ACGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCGTG CACCGCAGGGGTGAACTCGGCGGGGAGGTGGGCGGCCA	2849
	CAAGTTCCTGGCTTCTG	2850
	CAGAAGCCAGGAACTTG	2851
Haemoglobin variant Arg141Leu CGT-CTT	TGCACGCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCG TGCTGACCTCCAAATACCGTTAAGCTGGAGCCTCGGTGGCCA TGCTTCTTGCCCCTTGGGCCTCCCCCCAGCCCCTCCT	2852
	AGGAGGGGCTGGGGGGAGGCCCAAGGGGCAAGAAGCATGG CCACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAGCACG GTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCGTGCA	2853
	CAAATACCGTTAAGCTG	2854
	CAGCTTAACGGTATTTG	2855

EXAMPLE 16

Alpha-thalassemia - Hemoglobin alpha locus 2

The attached table discloses the correcting oligonucleotide base sequences for the hemoglobin alpha locus 2 oligonucleotides of the invention.

Table 23
HBA2 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia alpha Met(-1)Thr ATG-ACG	CCTGGCGCGCTCGCGGGCCGGCACTCTTCTGGTCCCCACAG ACTCAGAGAGAACCCACCATGGTGCTGTCTCCTGCCGACAAG ACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCA	2856
	TGCGCGCCGACCTTACCCCAGGCGGCCTTGACGTTGGTCTT GTCGGCAGGAGACAGCACCATGGTGGGTTCTCTCTGAGTCT GTGGGGACCAGAAGAGTGCCGGCCCGCGAGCGCGCCAGG	2857
	ACCCACCATGGTGCTGT	2858
	ACAGCACCATGGTGGGT	2859
Haemoglobin variant Ala12Asp GCC-GAC	CACAGACTCAGAGAGAACCCACCATGGTGCTGTCTCCTGCC GACAAGACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGC GCACGCTGGCGAGTATGGTGCGGAGGCCCTGGAGAGGTG	2860
	CACCTCTCCAGGGCCTCCGCACCATACTCGCCAGCGTGCGC GCCGACCTTACCCCAGGCGGCCTTGACGTTGGTCTTGTCGG CAGGAGACAGCACCATGGTGGGTTCTCTCTGAGTCTGTG	2861
	CGTCAAGGCCGCCTGGG	2862
	CCCAGGCGGCCTTGACG	2863
Haemoglobin variant Lys16Glu tAAG-GAG	AGAGAACCCACCATGGTGCTGTCTCCTGCCGACAAGACCAAC GTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCACGCTGGCG AGTATGGTGCGGAGGCCCTGGAGAGGTGAGGCTCCCTCC	2864
	GGAGGGAGCCTCACCTCTCCAGGGCCTCCGCACCATACTCG CCAGCGTGCGCGCCGACCTTACCCCAGGCGGCCTTGACGTT GGTCTTGTCGGCAGGAGACAGCACCATGGTGGGTTCTCT	2865
	CCTGGGGTAAGGTCGGC	2866
	GCCGACCTTACCCCAGG	2867
Haemoglobin variant His20Gln CACg-CAA	GGTGCTGTCTCCTGCCGACAAGACCAACGTCAAGGCCGCCT GGGGTAAGGTCGGCGCGCACGCTGGCGAGTATGGTGCGGA GGCCCTGGAGAGGTGAGGCTCCCTCCCCTGCTCCGACCCG	2868
	CGGGTCGGAGCAGGGGAGGGAGCCTCACCTCTCCAGGGCC TCCGCACCATACTCGCCAGCGTGCGCGCCGACCTTACCCCA GGCGGCCTTGACGTTGGTCTTGTCGGCAGGAGACAGCACC	2869
	GGCGCGCACGCTGGCGA	2870
	TCGCCAGCGTGCGCGCC	2871
Haemoglobin variant Glu27Asp GAGg-GAC	GACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCAC GCTGGCGAGTATGGTGCGGAGGCCCTGGAGAGGTGAGGCT CCCTCCCCTGCTCCGACCCGGGCTCCTCGCCCGCCCGGAC C	2872
	GGTCCGGGCGGGCGAGGAGCCCGGGTCGGAGCAGGGGAG GGAGCCTCACCTCTCCAGGGCCTCCGCACCATACTCGCCAG CGTGCGCGCCGACCTTACCCCAGGCGGCCTTGACGTTGGTC	2873
	GGTGCGGAGGCCCTGGA	2874
	TCCAGGGCCTCCGCACC	2875

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia alpha Leu29Pro CTG-CCG	ACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCACGCTGG CGAGTATGGTGCGGAGGCCCTGGAGAGGTGAGGCTCCCTCC CCTGCTCCGACCCGGGCTCCTCGCCCGCCCGGACCCACAG	2876
	CTGTGGGTCCGGGCGGGCGAGGAGCCCGGGTCGGAGCAGG GGAGGGAGCCTCACCTCTCCAGGGCCTCCGCACCATACTCG CCAGCGTGCGCGCCGACCTTACCCCAGGCGGCCTTGACGT	2877
	GGAGGCCCTGGAGAGGT	2878
	ACCTCTCCAGGGCCTCC	2879
Haemoglobin variant Asp47His cGAC-CAC	GCTTCTCCCCGCAGGATGTTCCTGTCCTTCCCCACCACCAAG ACCTACTTCCCGCACTTCGACCTGAGCCACGGCTCTGCCCA GGTTAAGGGCCACGGCAAGAAGGTGGCCGACGCGCTGA	2880
	TCAGCGCGTCGGCCACCTTCTTGCCGTGGCCCTTAACCTGG GCAGAGCCGTGGCTCAGGTCGAAGTGCGGGAAGTAGGTCTT GGTGGTGGGGAAGGACAGGAACATCCTGCGGGGAGAAGC	2881
	CGCACTTCGACCTGAGC	2882
	GCTCAGGTCGAAGTGCG	2883
Haemoglobin variant Leu48Arg CTG-CGG	CTCCCCGCAGGATGTTCCTGTCCTTCCCCACCACCAAGACCT ACTTCCCGCACTTCGACCTGAGCCACGGCTCTGCCCAGGTTA AGGGCCACGGCAAGAAGGTGGCCGACGCGCTGACCAA	2884
	TTGGTCAGCGCGTCGGCCACCTTCTTGCCGTGGCCCTTAAC CTGGGCAGAGCCGTGGCTCAGGTCGAAGTGCGGGAAGTAG GTCTTGGTGGTGGGGAAGGACAGGAACATCCTGCGGGGAG	2885
	CTTCGACCTGAGCCACG	2886
	CGTGGCTCAGGTCGAAG	2887
Haemoglobin variant Gln54Glu cCAG-GAG	CTGTCCTTCCCCACCACCAAGACCTACTTCCCGCACTTCGAC CTGAGCCACGGCTCTGCCCAGGTTAAGGGCCACGGCAAGAA GGTGGCCGACGCGCTGACCAACGCCGTGGCGCACGTGG	2888
	CCACGTGCGCCACGGCGTTGGTCAGCGCGTCGGCCACCTTC TTGCCGTGGCCCTTAACCTGGGCAGAGCCGTGGCTCAGGTC GAAGTGCGGGAAGTAGGTCTTGGTGGTGGGGAAGGACAG	2889
	GCTCTGCCCAGGTTAAG	2890
	CTTAACCTGGGCAGAGC	2891
Haemoglobin variant Gly59Asp GGC-GAC	CCAAGACCTACTTCCCGCACTTCGACCTGAGCCACGGCTCTG CCCAGGTTAAGGGCCACGGCAAGAAGGTGGCCGACGCGCT GACCAACGCCGTGGCGCACGTGGACGACATGCCCAACGC	2892
	GCGTTGGGCATGTCGTCCACGTGCGCCACGGCGTTGGTCAG CGCGTCGGCCACCTTCTTGCCGTGGCCCTTAACCTGGGCAG AGCCGTGGCTCAGGTCGAAGTGCGGGAAGTAGGTCTTGG	2893
	GGGCCACGGCAAGAAGG	2894
	CCTTCTTGCCGTGGCCC	2895
Haemoglobin variant Asn68Lys AACg-AAG	GAGCCACGGCTCTGCCCAGGTTAAGGGCCACGGCAAGAAGG TGGCCGACGCGCTGACCAACGCCGTGGCGCACGTGGACGA CATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCACGCG	2896

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CGCGTGCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATG TCGTCCACGTGCGCCACGGC**G**TTGGTCAGCGCGTCGGCCAC CTTCTTGCCGTGGCCCTTAACCTGGGCAGAGCCGTGGCTC	2897
	CTGACCAA**C**GCCGTGGC	2898
	GCCACGGC**G**TTGGTCAG	2899
Haemoglobin variant Asn68Lys AACg-AAA	GAGCCACGGCTCTGCCCAGGTTAAGGGCCACGGCAAGAAGG TGGCCGACGCGCTGACCAA**C**GCCGTGGCGCACGTGGACGA CATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCACGCG	2900
	CGCGTGCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATG TCGTCCACGTGCGCCACGGC**G**TTGGTCAGCGCGTCGGCCAC CTTCTTGCCGTGGCCCTTAACCTGGGCAGAGCCGTGGCTC	2901
	CTGACCAA**C**GCCGTGGC	2902
	GCCACGGC**G**TTGGTCAG	2903
Haemoglobin variant Asn78Lys AACg-AAA	CGGCAAGAAGGTGGCCGACGCGCTGACCAACGCCGTGGCG CACGTGGACGACATGCCCAA**C**GCGCTGTCCGCCCTGAGCGA CCTGCACGCGCACAAGCTTCGGGTGGACCCGGTCAACTTC	2904
	GAAGTTGACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGT CGCTCAGGGCGGACAGCGC**G**TTGGGCATGTCGTCCACGTGC GCCACGGCGTTGGTCAGCGCGTCGGCCACCTTCTTGCCG	2905
	ATGCCCAA**C**GCGCTGTC	2906
	GACAGCGC**G**TTGGGCAT	2907
Haemoglobin variant Asp85Val GAC-GTC	CGCTGACCAACGCCGTGGCGCACGTGGACGACATGCCCAAC GCGCTGTCCGCCCTGAGCG**A**CCTGCACGCGCACAAGCTTCG GGTGGACCCGGTCAACTTCAAGGTGAGCGGCGGGCCGGG	2908
	CCCGGCCCGCCGCTCACCTTGAAGTTGACCGGGTCCACCCG AAGCTTGTGCGCGTGCAGG**T**CGCTCAGGGCGGACAGCGCGT TGGGCATGTCGTCCACGTGCGCCACGGCGTTGGTCAGCG	2909
	CCTGAGCG**A**CCTGCACG	2910
	CGTGCAGG**T**CGCTCAGG	2911
Haemoglobin variant Lys90Asn AAGc-AAT	GGCGCACGTGGACGACATGCCCAACGCGCTGTCCGCCCTGA GCGACCTGCACGCGCACAA**G**CTTCGGGTGGACCCGGTCAAC TTCAAGGTGAGCGGCGGGCCGGGAGCGATCTGGGTCGAG	2912
	CTCGACCCAGATCGCTCCCGGCCCGCCGCTCACCTTGAAGT TGACCGGGTCCACCCGAAG**C**TTGTGCGCGTGCAGGTCGCTC AGGGCGGACAGCGCGTTGGGCATGTCGTCCACGTGCGCC	2913
	GCGCACAA**G**CTTCGGGT	2914
	ACCCGAAG**C**TTGTGCGC	2915
Haemoglobin variant Asp94His gGAC-CAC	GACGACATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCA CGCGCACAAGCTTCGGGTG**G**ACCCGGTCAACTTCAAGGTGA GCGGCGGGCCGGGAGCGATCTGGGTCGAGGGGCGAGATG	2916
	CATCTCGCCCCTCGACCCAGATCGCTCCCGGCCCGCCGCTC ACCTTGAAGTTGACCGGGT**C**CACCCGAAGCTTGTGCGCGTG CAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGTCGTC	2917
	TTCGGGTG**G**ACCCGGTC	2918

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GACCGGGTCCACCCGAA	2919
Haemoglobin variant Pro95Leu CCG-CTG	ACATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCACGCG CACAAGCTTCGGGTGGACCCGGTCAACTTCAAGGTGAGCGG CGGGCCGGGAGCGATCTGGGTCGAGGGGCGAGATGGCGC	2920
	GCGCCATCTCGCCCCTCGACCCAGATCGCTCCCGGCCCGCC GCTCACCTTGAAGTTGACCGGGTCCACCCGAAGCTTGTGCG CGTGCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGT	2921
	GGTGGACCCGGTCAACT	2922
	AGTTGACCGGGTCCACC	2923
Haemoglobin variant Ser102Arg aAGC-CGC	TAGCGCAGGCGGCGGCTGCGGGCCTGGGCCGCACTGACCC TCTTCTCTGCACAGCTCCTAAGCCACTGCCTGCTGGTGACCC TGGCCGCCCACCTCCCCGCCGAGTTCACCCCTGCGGTGC	2924
	GCACCGCAGGGGTGAACTCGGCGGGGAGGTGGGCGGCCAG GGTCACCAGCAGGCAGTGGCTTAGGAGCTGTGCAGAGAAGA GGGTCAGTGCGGCCCAGGCCCGCAGCCGCCGCCTGCGCTA	2925
	AGCTCCTAAGCCACTGC	2926
	GCAGTGGCTTAGGAGCT	2927
Haemoglobin H disease Cys104Tyr TGC-TAC	GGCGGCGGCTGCGGGCCTGGGCCGCACTGACCCTCTTCTCT GCACAGCTCCTAAGCCACTGCCTGCTGGTGACCCTGGCCGC CCACCTCCCCGCCGAGTTCACCCCTGCGGTGCACGCCTC	2928
	GAGGCGTGCACCGCAGGGGTGAACTCGGCGGGGAGGTGGG CGGCCAGGGTCACCAGCAGGCAGTGGCTTAGGAGCTGTGCA GAGAAGAGGGTCAGTGCGGCCCAGGCCCGCAGCCGCCGCC	2929
	AAGCCACTGCCTGCTGG	2930
	CCAGCAGGCAGTGGCTT	2931
Haemoglobin variant Ala111Val GCC-GTC	CCGCACTGACCCTCTTCTCTGCACAGCTCCTAAGCCACTGCC TGCTGGTGACCCTGGCCGCCCACCTCCCCGCCGAGTTCACC CCTGCGGTGCACGCCTCCCTGGACAAGTTCCTGGCTTC	2932
	GAAGCCAGGAACTTGTCCAGGGAGGCGTGCACCGCAGGGGT GAACTCGGCGGGGAGGTGGGCGGCCAGGGTCACCAGCAGG CAGTGGCTTAGGAGCTGTGCAGAGAAGAGGGTCAGTGCGG	2933
	CCTGGCCGCCCACCTCC	2934
	GGAGGTGGGCGGCCAGG	2935
Haemoglobin variant Ala120Glu GCG-GAG	TCCTAAGCCACTGCCTGCTGGTGACCCTGGCCGCCCACCTC CCCGCCGAGTTCACCCCTGCGGTGCACGCCTCCCTGGACAA GTTCCTGGCTTCTGTGAGCACCGTGCTGACCTCCAAATA	2936
	TATTTGGAGGTCAGCACGGTGCTCACAGAAGCCAGGAACTTG TCCAGGGAGGCGTGCACCGCAGGGGTGAACTCGGCGGGGA GGTGGGCGGCCAGGGTCACCAGCAGGCAGTGGCTTAGGA	2937
	CACCCCTGCGGTGCACG	2938
	CGTGCACCGCAGGGGTG	2939
Haemoglobin variant His122Gln CACg-CAG	CCACTGCCTGCTGGTGACCCTGGCCGCCCACCTCCCCGCCG AGTTCACCCCTGCGGTGCACGCCTCCCTGGACAAGTTCCTG GCTTCTGTGAGCACCGTGCTGACCTCCAAATACCGTTAA	2940

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTAACGGTATTTGGAGGTCAGCACGGTGCTCACAGAAGCCAG GAACTTGTCCAGGGAGGCGTGCACCGCAGGGGTGAACTCGG CGGGGAGGTGGGCGGCCAGGGTCACCAGCAGGCAGTGG	2941
	GCGGTGCACGCCTCCCT	2942
	AGGGAGGCGTGCACCGC	2943
Haemoglobin variant Ala123Ser cGCC-TCC	CACTGCCTGCTGGTGACCCTGGCCGCCCACCTCCCCGCCGA GTTCACCCCTGCGGTGCACGCCTCCCTGGACAAGTTCCTGG CTTCTGTGAGCACCGTGCTGACCTCCAAATACCGTTAAG	2944
	CTTAACGGTATTTGGAGGTCAGCACGGTGCTCACAGAAGCCA GGAACTTGTCCAGGGAGGCGTGCACCGCAGGGGTGAACTCG GCGGGGAGGTGGGCGGCCAGGGTCACCAGCAGGCAGTG	2945
	CGGTGCACGCCTCCCTG	2946
	CAGGGAGGCGTGCACCG	2947
Thalassaemia alpha Leu125Pro CTG-CCG	TGCTGGTGACCCTGGCCGCCCACCTCCCCGCCGAGTTCACC CCTGCGGTGCACGCCTCCCTGGACAAGTTCCTGGCTTCTGT GAGCACCGTGCTGACCTCCAAATACCGTTAAGCTGGAGC	2948
	GCTCCAGCTTAACGGTATTTGGAGGTCAGCACGGTGCTCACA GAAGCCAGGAACTTGTCCAGGGAGGCGTGCACCGCAGGGG TGAACTCGGCGGGGAGGTGGGCGGCCAGGGTCACCAGCA	2949
	CGCCTCCCTGGACAAGT	2950
	ACTTGTCCAGGGAGGCG	2951
Haemoglobin variant Ser131Pro tTCT-CCT	GCCCACCTCCCCGCCGAGTTCACCCCTGCGGTGCACGCCTC CCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTGCTGACCTC CAAATACCGTTAAGCTGGAGCCTCGGTAGCCGTTCCTC	2952
	GAGGAACGGCTACCGAGGCTCCAGCTTAACGGTATTTGGAG GTCAGCACGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGA GGCGTGCACCGCAGGGGTGAACTCGGCGGGGAGGTGGGC	2953
	TCCTGGCTTCTGTGAGC	2954
	GCTCACAGAAGCCAGGA	2955
Haemoglobin variant Leu136Met gCTG-ATG	GAGTTCACCCCTGCGGTGCACGCCTCCCTGGACAAGTTCCT GGCTTCTGTGAGCACCGTGCTGACCTCCAAATACCGTTAAGC TGGAGCCTCGGTAGCCGTTCCTCCTGCCCGCTGGGCCT	2956
	AGGCCCAGCGGGCAGGAGGAACGGCTACCGAGGCTCCAGC TTAACGGTATTTGGAGGTCAGCACGGTGCTCACAGAAGCCAG GAACTTGTCCAGGGAGGCGTGCACCGCAGGGGTGAACTC	2957
	GCACCGTGCTGACCTCC	2958
	GGAGGTCAGCACGGTGC	2959
Haemoglobin variant Leu136Pro CTG-CCG	AGTTCACCCCTGCGGTGCACGCCTCCCTGGACAAGTTCCTG GCTTCTGTGAGCACCGTGCTGACCTCCAAATACCGTTAAGCT GGAGCCTCGGTAGCCGTTCCTCCTGCCCGCTGGGCCTC	2960
	GAGGCCCAGCGGGCAGGAGGAACGGCTACCGAGGCTCCAG CTTAACGGTATTTGGAGGTCAGCACGGTGCTCACAGAAGCCA GGAACTTGTCCAGGGAGGCGTGCACCGCAGGGGTGAACT	2961
	CACCGTGCTGACCTCCA	2962

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGGAGGTCAGCACGGTG	2963
Haemoglobin variant Arg141Cys cCGT-TGT	GTGCACGCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACC GTGCTGACCTCCAAATACCGTTAAGCTGGAGCCTCGGTAGCC GTTCCTCCTGCCCGCTGGGCCTCCCAACGGGCCCTCC	2964
	GGAGGGCCCGTTGGGAGGCCCAGCGGGCAGGAGGAACGGC TACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAGCACGGT GCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCGTGCAC	2965
	CCAAATACCGTTAAGCT	2966
	AGCTTAACGGTATTTGG	2967
Haemoglobin variant Term142Gln tTAA-CAA	CACGCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTG CTGACCTCCAAATACCGTTAAGCTGGAGCCTCGGTAGCCGTT CCTCCTGCCCGCTGGGCCTCCCAACGGGCCCTCCTCC	2968
	GGAGGAGGGCCCGTTGGGAGGCCCAGCGGGCAGGAGGAAC GGCTACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAGCA CGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCGTG	2969
	AATACCGTTAAGCTGGA	2970
	TCCAGCTTAACGGTATT	2971
Haemoglobin variant Term142Lys tTAA-AAA	CACGCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTG CTGACCTCCAAATACCGTTAAGCTGGAGCCTCGGTAGCCGTT CCTCCTGCCCGCTGGGCCTCCCAACGGGCCCTCCTCC	2972
	GGAGGAGGGCCCGTTGGGAGGCCCAGCGGGCAGGAGGAAC GGCTACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAGCA CGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCGTG	2973
	AATACCGTTAAGCTGGA	2974
	TCCAGCTTAACGGTATT	2975
Haemoglobin variant Term142Tyr TAAg-TAT	CGCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTGCT GACCTCCAAATACCGTTAAGCTGGAGCCTCGGTAGCCGTTCC TCCTGCCCGCTGGGCCTCCCAACGGGCCCTCCTCCCC	2976
	GGGGAGGAGGGCCCGTTGGGAGGCCCAGCGGGCAGGAGG AACGGCTACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAG CACGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCG	2977
	TACCGTTAAGCTGGAGC	2978
	GCTCCAGCTTAACGGTA	2979

EXAMPLE 17
Human mismatch repair - MLH1

The human MLH1 gene is homologous to the bacterial *mutL* gene, which is involved in mismatch repair. Mutations in the MLH1 gene have been identified in many individuals with hereditary nonpolyposis colorectal cancer (HNPCC). Mutations in the MLH1 gene are also implicated in predisposition to a variety of cancers associated with, for example, Muir-Torre syndrome and Turcot

syndrome. The attached table discloses the correcting oligonucleotide base sequences for the MLH1 oligonucleotides of the invention.

Table 24
MLH1 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non-polyposis colorectal cancer Met1Arg ATG-AGG	TTGGCTGAAGGCACTTCCGTTGAGCATCTAGACGTTTCCTTG GCTCTTCTGGCGCCAAAA**T**GTCGTTCGTGGCAGGGGTTATTC GGCGGCTGGACGAGACAGTGGTGAACCGCATCGCGGC	2980
	GCCGCGATGCGGTTCACCACTGTCTCGTCCAGCCGCCGAAT AACCCCTGCCACGAACGAC**A**TTTTGGCGCCAGAAGAGCCAA GGAAACGTCTAGATGCTCAACGGAAGTGCCTTCAGCCAA	2981
	CGCCAAAA**T**GTCGTTCG	2982
	CGAACGAC**A**TTTTGGCG	2983
Non-polyposis colorectal cancer Met1Lys ATG-AAG	TTGGCTGAAGGCACTTCCGTTGAGCATCTAGACGTTTCCTTG GCTCTTCTGGCGCCAAAA**T**GTCGTTCGTGGCAGGGGTTATTC GGCGGCTGGACGAGACAGTGGTGAACCGCATCGCGGC	2984
	GCCGCGATGCGGTTCACCACTGTCTCGTCCAGCCGCCGAAT AACCCCTGCCACGAACGAC**A**TTTTGGCGCCAGAAGAGCCAA GGAAACGTCTAGATGCTCAACGGAAGTGCCTTCAGCCAA	2985
	CGCCAAAA**T**GTCGTTCG	2986
	CGAACGAC**A**TTTTGGCG	2987
Non-polyposis colorectal cancer Met35Arg ATG-AGG	TGGTGAACCGCATCGCGGCGGGGGAAGTTATCCAGCGGCCA GCTAATGCTATCAAAGAGA**T**GATTGAGAACTGGTACGGAGGG AGTCGAGCCGGGCTCACTTAAGGGCTACGACTTAACGG	2988
	CCGTTAAGTCGTAGCCCTTAAGTGAGCCCGGCTCGACTCCCT CCGTACCAGTTCTCAATC**A**TCTCTTTGATAGCATTAGCTGGCC GCTGGATAACTTCCCCCGCCGCGATGCGGTTCACCA	2989
	CAAAGAGA**T**GATTGAGA	2990
	TCTCAATC**A**TCTCTTTG	2991
Non-polyposis colorectal cancer Ser44Phe TCC-TTC	TAGAGTAGTTGCAGACTGATAAATTATTTTCTGTTTGATTTGCC AGTTTAGATGCAAAAT**C**CACAAGTATTCAAGTGATTGTTAAAG AGGGAGGCCTGAAGTTGATTCAGATCCAAGACAA	2992
	TTGTCTTGGATCTGAATCAACTTCAGGCCTCCCTCTTTAACAA TCACTTGAATACTTGTG**G**ATTTTGCATCTAAACTGGCAAATCA AACAGAAAATAATTTATCAGTCTGCAACTACTCTA	2993
	TGCAAAAT**C**CACAAGTA	2994
	TACTTGTG**G**ATTTTGCA	2995
Non-polyposis colorectal cancer Gln62Lys CAA-AAA	GCAAAATCCACAAGTATTCAAGTGATTGTTAAAGAGGGAGGC CTGAAGTTGATTCAGATC**C**AAGACAATGGCACCGGGATCAGG GTAAGTAAAACCTCAAAGTAGCAGGATGTTTGTGCGC	2996

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCGCACAAACATCCTGCTACTTTGAGGTTTTACTTACCCTGAT CCCGGTGCCATTGTCTTGGATCTGAATCAACTTCAGGCCTCC CTCTTTAACAATCACTTGAATACTTGTGGATTTTGC	2997
	TTCAGATCCAAGACAAT	2998
	ATTGTCTTGGATCTGAA	2999
Non-polyposis colorectal cancer Gln62Term CAA-TAA	GCAAAATCCACAAGTATTCAAGTGATTGTTAAAGAGGGAGGC CTGAAGTTGATTCAGATCCAAGACAATGGCACCGGGATCAGG GTAAGTAAAACCTCAAAGTAGCAGGATGTTTGTGCGC	3000
	GCGCACAAACATCCTGCTACTTTGAGGTTTTACTTACCCTGAT CCCGGTGCCATTGTCTTGGATCTGAATCAACTTCAGGCCTCC CTCTTTAACAATCACTTGAATACTTGTGGATTTTGC	3001
	TTCAGATCCAAGACAAT	3002
	ATTGTCTTGGATCTGAA	3003
Non-polyposis colorectal cancer Asn64Ser AAT-AGT	CCACAAGTATTCAAGTGATTGTTAAAGAGGGAGGCCTGAAGT TGATTCAGATCCAAGACAATGGCACCGGGATCAGGGTAAGTA AAACCTCAAAGTAGCAGGATGTTTGTGCGCTTCATGG	3004
	CCATGAAGCGCACAAACATCCTGCTACTTTGAGGTTTTACTTA CCCTGATCCCGGTGCCATTGTCTTGGATCTGAATCAACTTCA GGCCTCCCTCTTTAACAATCACTTGAATACTTGTGG	3005
	CCAAGACAATGGCACCG	3006
	CGGTGCCATTGTCTTGG	3007
Non-polyposis colorectal cancer Gly67Arg GGG-AGG	ATTCAAGTGATTGTTAAAGAGGGAGGCCTGAAGTTGATTCAGA TCCAAGACAATGGCACCGGGATCAGGGTAAGTAAAACCTCAA AGTAGCAGGATGTTTGTGCGCTTCATGGAAGAGTCA	3008
	TGACTCTTCCATGAAGCGCACAAACATCCTGCTACTTTGAGGT TTTACTTACCCTGATCCCGGTGCCATTGTCTTGGATCTGAATC AACTTCAGGCCTCCCTCTTTAACAATCACTTGAAT	3009
	ATGGCACCGGGATCAGG	3010
	CCTGATCCCGGTGCCAT	3011
Non-polyposis colorectal cancer Gly67Arg GGG-CGG	ATTCAAGTGATTGTTAAAGAGGGAGGCCTGAAGTTGATTCAGA TCCAAGACAATGGCACCGGGATCAGGGTAAGTAAAACCTCAA AGTAGCAGGATGTTTGTGCGCTTCATGGAAGAGTCA	3012
	TGACTCTTCCATGAAGCGCACAAACATCCTGCTACTTTGAGGT TTTACTTACCCTGATCCCGGTGCCATTGTCTTGGATCTGAATC AACTTCAGGCCTCCCTCTTTAACAATCACTTGAAT	3013
	ATGGCACCGGGATCAGG	3014
	CCTGATCCCGGTGCCAT	3015
Non-polyposis colorectal cancer Gly67Trp GGG-TGG	ATTCAAGTGATTGTTAAAGAGGGAGGCCTGAAGTTGATTCAGA TCCAAGACAATGGCACCGGGATCAGGGTAAGTAAAACCTCAA AGTAGCAGGATGTTTGTGCGCTTCATGGAAGAGTCA	3016
	TGACTCTTCCATGAAGCGCACAAACATCCTGCTACTTTGAGGT TTTACTTACCCTGATCCCGGTGCCATTGTCTTGGATCTGAATC AACTTCAGGCCTCCCTCTTTAACAATCACTTGAAT	3017
	ATGGCACCGGGATCAGG	3018

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCTGATCCCGGTGCCAT	3019
Non-polyposis colorectal cancer Cys77Arg TGT-CGT	GTAACATGATTATTTACTCATCTTTTTGGTATCTAACAGAAAGA AGATCTGGATATTGTATGTGAAAGGTTCACTACTAGTAAACTG CAGTCCTTTGAGGATTTAGCCAGTATTTCTACCT	3020
	AGGTAGAAATACTGGCTAAATCCTCAAAGGACTGCAGTTTACT AGTAGTGAACCTTTCACATACAATATCCAGATCTTCTTTCTGTT AGATACCAAAAAGATGAGTAAATAATCATGTTAC	3021
	ATATTGTATGTGAAAGG	3022
	CCTTTCACATACAATAT	3023
Non-polyposis colorectal cancer Cys77Tyr TGT-TAT	TAACATGATTATTTACTCATCTTTTTGGTATCTAACAGAAAGAA GATCTGGATATTGTATGTGAAAGGTTCACTACTAGTAAACTGC AGTCCTTTGAGGATTTAGCCAGTATTTCTACCTA	3024
	TAGGTAGAAATACTGGCTAAATCCTCAAAGGACTGCAGTTTAC TAGTAGTGAACCTTTCACATACAATATCCAGATCTTCTTTCTGT TAGATACCAAAAAGATGAGTAAATAATCATGTTA	3025
	TATTGTATGTGAAAGGT	3026
	ACCTTTCACATACAATA	3027
Non-polyposis colorectal cancer Ser93Gly AGT-GGT	CTGGATATTGTATGTGAAAGGTTCACTACTAGTAAACTGCAGT CCTTTGAGGATTTAGCCAGTATTTCTACCTATGGCTTTCGAGG TGAGGTAAGCTAAAGATTCAAGAAATGTGTAAAAT	3028
	ATTTTACACATTTCTTGAATCTTTAGCTTACCTCACCTCGAAAG CCATAGGTAGAAATACTGGCTAAATCCTCAAAGGACTGCAGTT TACTAGTAGTGAACCTTTCACATACAATATCCAG	3029
	ATTTAGCCAGTATTTCT	3030
	AGAAATACTGGCTAAAT	3031
Non-polyposis colorectal cancer Arg100Term CGA-TGA	TTCACTACTAGTAAACTGCAGTCCTTTGAGGATTTAGCCAGTA TTTCTACCTATGGCTTTCGAGGTGAGGTAAGCTAAAGATTCAA GAAATGTGTAAAATATCCTCCTGTGATGACATTGT	3032
	ACAATGTCATCACAGGAGGATATTTTACACATTTCTTGAATCTT TAGCTTACCTCACCTCGAAAGCCATAGGTAGAAATACTGGCTA AATCCTCAAAGGACTGCAGTTTACTAGTAGTGAA	3033
	ATGGCTTTCGAGGTGAG	3034
	CTCACCTCGAAAGCCAT	3035
Non-polyposis colorectal cancer Ile107Arg ATA-AGA	ACCCAGCAGTGAGTTTTTCTTTCAGTCTATTTTCTTTTCTTCCT TAGGCTTTGGCCAGCATAAGCCATGTGGCTCATGTTACTATTA CAACGAAAACAGCTGATGGAAAGTGTGCATACAG	3036
	CTGTATGCACACTTTCCATCAGCTGTTTTCGTTGTAATAGTAA CATGAGCCACATGGCTTATGCTGGCCAAAGCCTAAGGAAGAA AAGAAAATAGACTGAAAGAAAAACTCACTGCTGGGT	3037
	GGCCAGCATAAGCCATG	3038
	CATGGCTTATGCTGGCC	3039

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non-polyposis colorectal cancer Thr117Arg ACG-AGG	TTTCTTTTCTTCCTTAGGCTTTGGCCAGCATAAGCCATGTGGC TCATGTTACTATTACAACGAAAACAGCTGATGGAAAGTGTGCA TACAGGTATAGTGCTGACTTCTTTTACTCATATAT	3040
	ATATATGAGTAAAAGAAGTCAGCACTATACCTGTATGCACACT TTCCATCAGCTGTTTTCGTTGTAATAGTAACATGAGCCACATG GCTTATGCTGGCCAAAGCCTAAGGAAGAAAAGAAA	3041
	TATTACAACGAAAACAG	3042
	CTGTTTTCGTTGTAATA	3043
Non-polyposis colorectal cancer Thr117Met ACG-ATG	TTTCTTTTCTTCCTTAGGCTTTGGCCAGCATAAGCCATGTGGC TCATGTTACTATTACAACGAAAACAGCTGATGGAAAGTGTGCA TACAGGTATAGTGCTGACTTCTTTTACTCATATAT	3044
	ATATATGAGTAAAAGAAGTCAGCACTATACCTGTATGCACACT TTCCATCAGCTGTTTTCGTTGTAATAGTAACATGAGCCACATG GCTTATGCTGGCCAAAGCCTAAGGAAGAAAAGAAA	3045
	TATTACAACGAAAACAG	3046
	CTGTTTTCGTTGTAATA	3047
Non-polyposis colorectal cancer Gly133Term GGA-TGA	TCTATCTCTCTACTGGATATTAATTTGTTATATTTTCTCATTAGA GCAAGTTACTCAGATGGAAAACTGAAAGCCCCTCCTAAACCA TGTGCTGGCAATCAAGGGACCCAGATCACGGTAA	3048
	TTACCGTGATCTGGGTCCCTTGATTGCCAGCACATGGTTTAG GAGGGGCTTTCAGTTTTCCATCTGAGTAACTTGCTCTAATGAG AAAATATAACAAATTAATATCCAGTAGAGAGATAGA	3049
	ACTCAGATGGAAAACTG	3050
	CAGTTTTCCATCTGAGT	3051
Non-polyposis colorectal cancer Val185Gly GTA-GGA	TAGTGTGTGTTTTTGGCAACTCTTTTCTTACTCTTTTGTTTTTC TTTTCCAGGTATTCAGTACACAATGCAGGCATTAGTTTCTCAG TTAAAAAAGTAAGTTCTTGGTTTATGGGGGATGG	3052
	CCATCCCCCATAAACCAAGAACTTACTTTTTTAACTGAGAAAC TAATGCCTGCATTGTGTACTGAATACCTGGAAAAGAAAAACAA AAGAGTAAGAAAAGAGTTGCCAAAAACACACACTA	3053
	GTATTCAGTACACAATG	3054
	CATTGTGTACTGAATAC	3055
Non-polyposis colorectal cancer Ser193Pro TCA-CCA	TTTCTTACTCTTTTGTTTTTCTTTTCCAGGTATTCAGTACACAAT GCAGGCATTAGTTTCTCAGTTAAAAAAGTAAGTTCTTGGTTTAT GGGGGATGGTTTTGTTTTATGAAAAGAAAAAA	3056
	TTTTTTCTTTTCATAAAACAAAACCATCCCCCATAAACCAAGAA CTTACTTTTTTAACTGAGAAACTAATGCCTGCATTGTGTACTG AATACCTGGAAAAGAAAAACAAAAGAGTAAGAAA	3057
	TTAGTTTCTCAGTTAAA	3058
	TTTAACTGAGAAACTAA	3059
Non-polyposis colorectal cancer Val213Met GTG-ATG	TTTGTTTATCAGCAAGGAGAGACAGTAGCTGATGTTAGGACA CTACCCAATGCCTCAACCGTGGACAATATTCGCTCCATCTTTG GAAATGCTGTTAGTCGGTATGTCGATAACCTATATA	3060

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TATATAGGTTATCGACATACCGACTAACAGCATTTCCAAAGAT GGAGCGAATATTGTCCACGGTTGAGGCATTGGGTAGTGTCCT AACATCAGCTACTGTCTCTCCTTGCTGATAAACAAA	3061
	CCTCAACCGTGGACAAT	3062
	ATTGTCCACGGTTGAGG	3063
Non-polyposis colorectal cancer Arg217Cys CGC-TGC	CAAGGAGAGACAGTAGCTGATGTTAGGACACTACCCAATGCC TCAACCGTGGACAATATTCGCTCCATCTTTGGAAATGCTGTTA GTCGGTATGTCGATAACCTATATAAAAAAATCTTTT	3064
	AAAAGATTTTTTTATATAGGTTATCGACATACCGACTAACAGCA TTTCCAAAGATGGAGCGAATATTGTCCACGGTTGAGGCATTG GGTAGTGTCCTAACATCAGCTACTGTCTCTCCTTG	3065
	ACAATATTCGCTCCATC	3066
	GATGGAGCGAATATTGT	3067
Non-polyposis colorectal cancer Ile219Val ATC-GTC	GAGACAGTAGCTGATGTTAGGACACTACCCAATGCCTCAACC GTGGACAATATTCGCTCCATCTTTGGAAATGCTGTTAGTCGGT ATGTCGATAACCTATATAAAAAAATCTTTTACATTT	3068
	AAATGTAAAAGATTTTTTTATATAGGTTATCGACATACCGACTA ACAGCATTTCCAAAGATGGAGCGAATATTGTCCACGGTTGAG GCATTGGGTAGTGTCCTAACATCAGCTACTGTCTC	3069
	TTCGCTCCATCTTTGGA	3070
	TCCAAAGATGGAGCGAA	3071
Non-polyposis colorectal cancer Gly244Asp GGT-GAT	CTAATAGAGAACTGATAGAAATTGGATGTGAGGATAAAACCCT AGCCTTCAAAATGAATGGTTACATATCCAATGCAAACTACTCA GTGAAGAAGTGCATCTTCTTACTCTTCATCAACCG	3072
	CGGTTGATGAAGAGTAAGAAGATGCACTTCTTCACTGAGTAG TTTGCATTGGATATGTAACCATTCATTTTGAAGGCTAGGGTTT TATCCTCACATCCAATTTCTATCAGTTCTCTATTAG	3073
	AATGAATGGTTACATAT	3074
	ATATGTAACCATTCATT	3075
Non-polyposis colorectal cancer Ser252Term TCA-TAA	GATGTGAGGATAAAACCCTAGCCTTCAAAATGAATGGTTACAT ATCCAATGCAAACTACTCAGTGAAGAAGTGCATCTTCTTACTC TTCATCAACCGTAAGTTAAAAAGAACCACATGGGA	3076
	TCCCATGTGGTTCTTTTTAACTTACGGTTGATGAAGAGTAAGA AGATGCACTTCTTCACTGAGTAGTTTGCATTGGATATGTAACC ATTCATTTTGAAGGCTAGGGTTTTATCCTCACATC	3077
	AAACTACTCAGTGAAGA	3078
	TCTTCACTGAGTAGTTT	3079
Non-polyposis colorectal cancer Glu268Gly GAA-GGA	CACCCCTCAGGACAGTTTTGAACTGGTTGCTTTCTTTTTATTG TTTAGATCGTCTGGTAGAATCAACTTCCTTGAGAAAAGCCATA GAAACAGTGTATGCAGCCTATTTGCCCAAAAACAC	3080
	GTGTTTTTGGGCAAATAGGCTGCATACACTGTTTCTATGGCTT TTCTCAAGGAAGTTGATTCTACCAGACGATCTAAACAATAAAA AGAAAGCAACCAGTTCAAAACTGTCCTGAGGGGTG	3081
	TCTGGTAGAATCAACTT	3082

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AAGTTGATTCTACCAGA	3083
Non-polyposis colorectal cancer Ser269Term TCA-TGA	CCCTCAGGACAGTTTTGAACTGGTTGCTTTCTTTTTATTGTTTA GATCGTCTGGTAGAATCAACTTCCTTGAGAAAAGCCATAGAAA CAGTGTATGCAGCCTATTTGCCCAAAAACACACA	3084
	TGTGTGTTTTTGGGCAAATAGGCTGCATACACTGTTTCTATGG CTTTTCTCAAGGAAGTTGATTCTACCAGACGATCTAAACAATA AAAAGAAAGCAACCAGTTCAAAACTGTCCTGAGGG	3085
	GGTAGAATCAACTTCCT	3086
	AGGAAGTTGATTCTACC	3087
Non-polyposis colorectal cancer Glu297Term GAA-TAA	CTTTTTCTCCCCCTCCCACTATCTAAGGTAATTGTTCTCTCTTA TTTTCCTGACAGTTTAGAAATCAGTCCCCAGAATGTGGATGTT AATGTGCACCCCACAAAGCATGAAGTTCACTTCC	3088
	GGAAGTGAACTTCATGCTTTGTGGGGTGCACATTAACATCCA CATTCTGGGGACTGATTTCTAAACTGTCAGGAAAATAAGAGAG AACAATTACCTTAGATAGTGGGAGGGGGAGAAAAAG	3089
	ACAGTTTAGAAATCAGT	3090
	ACTGATTTCTAAACTGT	3091
Non-polyposis colorectal cancer Gln301Term CAG-TAG	CTCCCACTATCTAAGGTAATTGTTCTCTCTTATTTTCCTGACAG TTTAGAAATCAGTCCCCAGAATGTGGATGTTAATGTGCACCCC ACAAAGCATGAAGTTCACTTCCTGCACGAGGAGA	3092
	TCTCCTCGTGCAGGAAGTGAACTTCATGCTTTGTGGGGTGCA CATTAACATCCACATTCTGGGGACTGATTTCTAAACTGTCAGG AAAATAAGAGAGAACAATTACCTTAGATAGTGGGAG	3093
	TCAGTCCCCAGAATGTG	3094
	CACATTCTGGGGACTGA	3095
Non-polyposis colorectal cancer Val326Ala GTG-GCG	ATGTGCACCCCACAAAGCATGAAGTTCACTTCCTGCACGAGG AGAGCATCCTGGAGCGGGTGCAGCAGCACATCGAGAGCAAG CTCCTGGGCTCCAATTCCTCCAGGATGTACTTCACCCA	3096
	TGGGTGAAGTACATCCTGGAGGAATTGGAGCCCAGGAGCTT GCTCTCGATGTGCTGCTGCACCCGCTCCAGGATGCTCTCCT CGTGCAGGAAGTGAACTTCATGCTTTGTGGGGTGCACAT	3097
	GGAGCGGGTGCAGCAGC	3098
	GCTGCTGCACCCGCTCC	3099
Non-polyposis colorectal cancer His329Pro CAC-CCC	CCACAAAGCATGAAGTTCACTTCCTGCACGAGGAGAGCATCC TGGAGCGGGTGCAGCAGCACATCGAGAGCAAGCTCCTGGGC TCCAATTCCTCCAGGATGTACTTCACCCAGGTCAGGGC	3100
	GCCCTGACCTGGGTGAAGTACATCCTGGAGGAATTGGAGCC CAGGAGCTTGCTCTCGATGTGCTGCTGCACCCGCTCCAGGA TGCTCTCCTCGTGCAGGAAGTGAACTTCATGCTTTGTGG	3101
	GCAGCAGCACATCGAGA	3102
	TCTCGATGTGCTGCTGC	3103

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non-polyposis colorectal cancer Val384Asp GTT-GAT	CAAGTCTGACCTCGTCTTCTACTTCTGGAAGTAGTGATAAGGT CTATGCCCACCAGATGGTTCGTACAGATTCCCGGGAACAGAA GCTTGATGCATTTCTGCAGCCTCTGAGCAAACCCCT	3104
	AGGGGTTTGCTCAGAGGCTGCAGAAATGCATCAAGCTTCTGT TCCCGGGAATCTGTACGAACCATCTGGTGGGCATAGACCTTA TCACTACTTCCAGAAGTAGAAGACGAGGTCAGACTTG	3105
	CCAGATGGTTCGTACAG	3106
	CTGTACGAACCATCTGG	3107
Non-polyposis colorectal cancer Ala441Thr GCT-ACT	AGTGGCAGGGCTAGGCAGCAAGATGAGGAGATGCTTGAACT CCCAGCCCCTGCTGAAGTGGCTGCCAAAAATCAGAGCTTGGA GGGGGATACAACAAAGGGGACTTCAGAAATGTCAGAGA	3108
	TCTCTGACATTTCTGAAGTCCCCTTTGTTGTATCCCCCTCCAA GCTCTGATTTTTGGCAGCCACTTCAGCAGGGGCTGGGAGTTC AAGCATCTCCTCATCTTGCTGCCTAGCCCTGCCACT	3109
	CTGAAGTGGCTGCCAAA	3110
	TTTGGCAGCCACTTCAG	3111
Non-polyposis colorectal cancer Arg487Term CGA-TGA	CTTCATTGCAGAAAGAGACATCGGGAAGATTCTGATGTGGAA ATGGTGGAAGATGATTCCCGAAAGGAAATGACTGCAGCTTGT ACCCCCCGGAGAAGGATCATTAACCTCACTAGTGTTT	3112
	AAACACTAGTGAGGTTAATGATCCTTCTCCGGGGGGTACAAG CTGCAGTCATTTCCTTTCGGGAATCATCTTCCACCATTTCCAC ATCAGAATCTTCCCGATGTCTCTTTCTGCAATGAAG	3113
	ATGATTCCCGAAAGGAA	3114
	TTCCTTTCGGGAATCAT	3115
Non-polyposis colorectal cancer Ala492Thr GCA-ACA	AGACATCGGGAAGATTCTGATGTGGAAATGGTGGAAGATGAT TCCCGAAAGGAAATGACTGCAGCTTGTACCCCCCGGAGAAG GATCATTAACCTCACTAGTGTTTTGAGTCTCCAGGAAG	3116
	CTTCCTGGAGACTCAAAACACTAGTGAGGTTAATGATCCTTCT CCGGGGGGTACAAGCTGCAGTCATTTCCTTTCGGGAATCATC TTCCACCATTTCCACATCAGAATCTTCCCGATGTCT	3117
	AAATGACTGCAGCTTGT	3118
	ACAAGCTGCAGTCATTT	3119
Non-polyposis colorectal cancer Val506Ala GTT-GCT	CCCGAAAGGAAATGACTGCAGCTTGTACCCCCCGGAGAAGG ATCATTAACCTCACTAGTGTTTTGAGTCTCCAGGAAGAAATTA ATGAGCAGGGACATGAGGGTACGTAAACGCTGTGGCC	3120
	GGCCACAGCGTTTACGTACCCTCATGTCCCTGCTCATTAATTT CTTCCTGGAGACTCAAAACACTAGTGAGGTTAATGATCCTTCT CCGGGGGGTACAAGCTGCAGTCATTTCCTTTCGGG	3121
	CACTAGTGTTTTGAGTC	3122
	GACTCAAAACACTAGTG	3123
Non-polyposis colorectal cancer Gln542Leu CAG-CTG	GGGAGATGTTGCATAACCACTCCTTCGTGGGCTGTGTGAATC CTCAGTGGGCCTTGGCACAGCATCAAACCAAGTTATACCTTC TCAACACCACCAAGCTTAGGTAAATCAGCTGAGTGTG	3124

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CACACTCAGCTGATTTACCTAAGCTTGGTGGTGTTGAGAAGG TATAACTTGGTTTGATGCTGTGCCAAGGCCCACTGAGGATTC ACACAGCCCACGAAGGAGTGGTTATGCAACATCTCCC	3125
	CTTGGCACAGCATCAAA	3126
	TTTGATGCTGTGCCAAG	3127
Non-polyposis colorectal cancer Leu549Pro CTT-CCT	CCTTCGTGGGCTGTGTGAATCCTCAGTGGGCCTTGGCACAG CATCAAACCAAGTTATACCTTCTCAACACCACCAAGCTTAGGT AAATCAGCTGAGTGTGTGAACAAGCAGAGCTACTACA	3128
	TGTAGTAGCTCTGCTTGTTCACACACTCAGCTGATTTACCTAA GCTTGGTGGTGTTGAGAAGGTATAACTTGGTTTGATGCTGTG CCAAGGCCCACTGAGGATTCACACAGCCCACGAAGG	3129
	GTTATACCTTCTCAACA	3130
	TGTTGAGAAGGTATAAC	3131
Non-polyposis colorectal cancer Asn551Thr AAC-ACC	TGGGCTGTGTGAATCCTCAGTGGGCCTTGGCACAGCATCAAA CCAAGTTATACCTTCTCAACACCACCAAGCTTAGGTAAATCAG CTGAGTGTGTGAACAAGCAGAGCTACTACAACAATG	3132
	CATTGTTGTAGTAGCTCTGCTTGTTCACACACTCAGCTGATTT ACCTAAGCTTGGTGGTGTTGAGAAGGTATAACTTGGTTTGATG CTGTGCCAAGGCCCACTGAGGATTCACACAGCCCA	3133
	CCTTCTCAACACCACCA	3134
	TGGTGGTGTTGAGAAGG	3135
Non-polyposis colorectal cancer Gln562Term CAG-TAG	ATGAATTCAGCTTTTCCTTAAAGTCACTTCATTTTTATTTTCAG TGAAGAACTGTTCTACCAGATACTCATTTATGATTTTGCCAATT TTGGTGTTCTCAGGTTATCGGTAAGTTTAGATC	3136
	GATCTAAACTTACCGATAACCTGAGAACACCAAAATTGGCAAA ATCATAAATGAGTATCTGGTAGAACAGTTCTTCACTGAAAATA AAAATGAAGTGACTTTAAGGAAAAGCTGAATTCAT	3137
	TGTTCTACCAGATACTC	3138
	GAGTATCTGGTAGAACA	3139
Non-polyposis colorectal cancer Ile565Phe ATT-TTT	GCTTTTCCTTAAAGTCACTTCATTTTTATTTTCAGTGAAGAACT GTTCTACCAGATACTCATTTATGATTTTGCCAATTTTGGTGTTC TCAGGTTATCGGTAAGTTTAGATCCTTTTCACT	3140
	AGTGAAAAGGATCTAAACTTACCGATAACCTGAGAACACCAAA ATTGGCAAAATCATAAATGAGTATCTGGTAGAACAGTTCTTCA CTGAAAATAAAAATGAAGTGACTTTAAGGAAAAGC	3141
	AGATACTCATTTATGAT	3142
	ATCATAAATGAGTATCT	3143
Non-polyposis colorectal cancer Leu574Pro CTC-CCC	TTTTCAGTGAAGAACTGTTCTACCAGATACTCATTTATGATTTT GCCAATTTTGGTGTTCTCAGGTTATCGGTAAGTTTAGATCCTT TTCACTTCTGAAATTTCAACTGATCGTTTCTGAA	3144
	TTCAGAAACGATCAGTTGAAATTTCAGAAGTGAAAAGGATCTA AACTTACCGATAACCTGAGAACACCAAAATTGGCAAAATCATA AATGAGTATCTGGTAGAACAGTTCTTCACTGAAAA	3145
	TGGTGTTCTCAGGTTAT	3146

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATAACCTGAGAACACCA	3147
Non-polyposis colorectal cancer Leu582Val CTC-GTC	TGGATGCTCCGTTAAAGCTTGCTCCTTCATGTTCTTGCTTCTT CCTAGGAGCCAGCACCGCTCTTTGACCTTGCCATGCTTGCCT TAGATAGTCCAGAGAGTGGCTGGACAGAGGAAGATG	3148
	CATCTTCCTCTGTCCAGCCACTCTCTGGACTATCTAAGGCAA GCATGGCAAGGTCAAAGAGCGGTGCTGGCTCCTAGGAAGAA GCAAGAACATGAAGGAGCAAGCTTTAACGGAGCATCCA	3149
	CAGCACCGCTCTTTGAC	3150
	GTCAAAGAGCGGTGCTG	3151
Non-polyposis colorectal cancer Leu607His CTT-CAT	TGCTTGCCTTAGATAGTCCAGAGAGTGGCTGGACAGAGGAAG ATGGTCCCAAAGAAGGACTTGCTGAATACATTGTTGAGTTTCT GAAGAAGAAGGCTGAGATGCTTGCAGACTATTTCTC	3152
	GAGAAATAGTCTGCAAGCATCTCAGCCTTCTTCTTCAGAAACT CAACAATGTATTCAGCAAGTCCTTCTTTGGGACCATCTTCCTC TGTCCAGCCACTCTCTGGACTATCTAAGGCAAGCA	3153
	AGAAGGACTTGCTGAAT	3154
	ATTCAGCAAGTCCTTCT	3155
Non-polyposis colorectal cancer Lys618Term AAG-TAG	ACAGAGGAAGATGGTCCCAAAGAAGGACTTGCTGAATACATT GTTGAGTTTCTGAAGAAGAAGGCTGAGATGCTTGCAGACTAT TTCTCTTTGGAAATTGATGAGGTGTGACAGCCATTCT	3156
	AGAATGGCTGTCACACCTCATCAATTTCCAAAGAGAAATAGTC TGCAAGCATCTCAGCCTTCTTCTTCAGAAACTCAACAATGTAT TCAGCAAGTCCTTCTTTGGGACCATCTTCCTCTGT	3157
	TGAAGAAGAAGGCTGAG	3158
	CTCAGCCTTCTTCTTCA	3159
Non-polyposis colorectal cancer Lys618Thr AAG-ACG	CAGAGGAAGATGGTCCCAAAGAAGGACTTGCTGAATACATTG TTGAGTTTCTGAAGAAGAAGGCTGAGATGCTTGCAGACTATTT CTCTTTGGAAATTGATGAGGTGTGACAGCCATTCTT	3160
	AAGAATGGCTGTCACACCTCATCAATTTCCAAAGAGAAATAGT CTGCAAGCATCTCAGCCTTCTTCTTCAGAAACTCAACAATGTA TTCAGCAAGTCCTTCTTTGGGACCATCTTCCTCTG	3161
	GAAGAAGAAGGCTGAGA	3162
	TCTCAGCCTTCTTCTTC	3163
Non-polyposis colorectal cancer Arg659Leu CGA-CTA	TACCCCTTCTGATTGACAACTATGTGCCCCCTTTGGAGGGAC TGCCTATCTTCATTCTTCGACTAGCCACTGAGGTCAGTGATCA AGCAGATACTAAGCATTTCGGTACATGCATGTGTGC	3164
	GCACACATGCATGTACCGAAATGCTTAGTATCTGCTTGATCAC TGACCTCAGTGGCTAGTCGAAGAATGAAGATAGGCAGTCCCT CCAAAGGGGGCACATAGTTGTCAATCAGAAGGGGTA	3165
	CATTCTTCGACTAGCCA	3166
	TGGCTAGTCGAAGAATG	3167

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non-polyposis colorectal cancer Arg659Pro CGA-CCA	TACCCCTTCTGATTGACAACTATGTGCCCCCTTTGGAGGGAC TGCCTATCTTCATTCTTCGACTAGCCACTGAGGTCAGTGATCA AGCAGATACTAAGCATTTCGGTACATGCATGTGTGC	3168
	GCACACATGCATGTACCGAAATGCTTAGTATCTGCTTGATCAC TGACCTCAGTGGCTAGTCGAAGAATGAAGATAGGCAGTCCCT CCAAAGGGGGCACATAGTTGTCAATCAGAAGGGGTA	3169
	CATTCTTCGACTAGCCA	3170
	TGGCTAGTCGAAGAATG	3171
Non-polyposis colorectal cancer Arg659Term CGA-TGA	TTACCCCTTCTGATTGACAACTATGTGCCCCCTTTGGAGGGA CTGCCTATCTTCATTCTTCGACTAGCCACTGAGGTCAGTGATC AAGCAGATACTAAGCATTTCGGTACATGCATGTGTG	3172
	CACACATGCATGTACCGAAATGCTTAGTATCTGCTTGATCACT GACCTCAGTGGCTAGTCGAAGAATGAAGATAGGCAGTCCCTC CAAAGGGGGCACATAGTTGTCAATCAGAAGGGGTAA	3173
	TCATTCTTCGACTAGCC	3174
	GGCTAGTCGAAGAATGA	3175
Non-polyposis colorectal cancer Ala681Thr GCT-ACT	TTGGACCAGGTGAATTGGGACGAAGAAAAGGAATGTTTTGAA AGCCTCAGTAAAGAATGCGCTATGTTCTATTCCATCCGGAAG CAGTACATATCTGAGGAGTCGACCCTCTCAGGCCAGC	3176
	GCTGGCCTGAGAGGGTCGACTCCTCAGATATGTACTGCTTCC GGATGGAATAGAACATAGCGCATTCTTTACTGAGGCTTTCAAA ACATTCCTTTTCTTCGTCCCAATTCACCTGGTCCAA	3177
	AAGAATGCGCTATGTTC	3178
	GAACATAGCGCATTCTT	3179
Non-polyposis colorectal cancer Trp712Term TGG-TAG	AGGCTTATGACATCTAATGTGTTTTCCAGAGTGAAGTGCCTGG CTCCATTCCAAACTCCTGGAAGTGGACTGTGGAACACATTGT CTATAAAGCCTTGCGCTCACACATTCTGCCTCCTAA	3180
	TTAGGAGGCAGAATGTGTGAGCGCAAGGCTTTATAGACAATG TGTTCCACAGTCCACTTCCAGGAGTTTGGAATGGAGCCAGGC ACTTCACTCTGGAAAACACATTAGATGTCATAAGCCT	3181
	AAACTCCTGGAAGTGGA	3182
	TCCACTTCCAGGAGTTT	3183
Non-polyposis colorectal cancer Trp714Term TGG-TAG	ATGACATCTAATGTGTTTTCCAGAGTGAAGTGCCTGGCTCCAT TCCAAACTCCTGGAAGTGGACTGTGGAACACATTGTCTATAAA GCCTTGCGCTCACACATTCTGCCTCCTAAACATTT	3184
	AAATGTTTAGGAGGCAGAATGTGTGAGCGCAAGGCTTTATAG ACAATGTGTTCCACAGTCCACTTCCAGGAGTTTGGAATGGAG CCAGGCACTTCACTCTGGAAAACACATTAGATGTCAT	3185
	CTGGAAGTGGACTGTGG	3186
	CCACAGTCCACTTCCAG	3187
Non-polyposis colorectal cancer Trp714Term TGG-TGA	TGACATCTAATGTGTTTTCCAGAGTGAAGTGCCTGGCTCCATT CCAAACTCCTGGAAGTGGACTGTGGAACACATTGTCTATAAA GCCTTGCGCTCACACATTCTGCCTCCTAAACATTTC	3188

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAAATGTTTAGGAGGCAGAATGTGTGAGCGCAAGGCTTTATA GACAATGTGTTCCACAGT**C**CACTTCCAGGAGTTTGGAATGGA GCCAGGCACTTCACTCTGGAAAACACATTAGATGTCA	3189
	TGGAAGTG**G**ACTGTGGA	3190
	TCCACAGT**C**CACTTCCA	3191
Non-polyposis colorectal cancer Val716Met GTG-ATG	ATCTAATGTGTTTTCCAGAGTGAAGTGCCTGGCTCCATTCCAA ACTCCTGGAAGTGGACT**G**TGGAACACATTGTCTATAAAGCCTT GCGCTCACACATTCTGCCTCCTAAACATTTCACAG	3192
	CTGTGAAATGTTTAGGAGGCAGAATGTGTGAGCGCAAGGCTT TATAGACAATGTGTTCCA**C**AGTCCACTTCCAGGAGTTTGGAAT GGAGCCAGGCACTTCACTCTGGAAAACACATTAGAT	3193
	AGTGGACT**G**TGGAACAC	3194
	GTGTTCCA**C**AGTCCACT	3195
Non-polyposis colorectal cancer Tyr721Term TAT-TAA	GAGTGAAGTGCCTGGCTCCATTCCAAACTCCTGGAAGTGGAC TGTGGAACACATTGTCTA**T**AAAGCCTTGCGCTCACACATTCTG CCTCCTAAACATTTCACAGAAGATGGAAATATCCTG	3196
	CAGGATATTTCCATCTTCTGTGAAATGTTTAGGAGGCAGAATG TGTGAGCGCAAGGCTTT**A**TAGACAATGTGTTCCACAGTCCAC TTCCAGGAGTTTGGAATGGAGCCAGGCACTTCACTC	3197
	ATTGTCTA**T**AAAGCCTT	3198
	AAGGCTTT**A**TAGACAAT	3199
Non-polyposis colorectal cancer Lys751Arg AAA-AGA	CTAAACATTTCACAGAAGATGGAAATATCCTGCAGCTTGCTAA CCTGCCTGATCTATACA**A**AGTCTTTGAGAGGTGTTAAATATGG TTATTTATGCACTGTGGGATGTGTTCTTCTTTCTC	3200
	GAGAAAGAAGAACACATCCCACAGTGCATAAATAACCATATTT AACACCTCTCAAAGACT**T**TGTATAGATCAGGCAGGTTAGCAAG CTGCAGGATATTTCCATCTTCTGTGAAATGTTTAG	3201
	TCTATACA**A**AGTCTTTG	3202
	CAAAGACT**T**TGTATAGA	3203
Non-polyposis colorectal cancer Arg755Trp AGG-TGG	ACAGAAGATGGAAATATCCTGCAGCTTGCTAACCTGCCTGAT CTATACAAAGTCTTTGAG**A**GGTGTTAAATATGGTTATTTATGCA CTGTGGGATGTGTTCTTCTTTCTCTGTATTCCGAT	3204
	ATCGGAATACAGAGAAAGAAGAACACATCCCACAGTGCATAA ATAACCATATTTAACACC**T**CTCAAAGACTTTGTATAGATCAGG CAGGTTAGCAAGCTGCAGGATATTTCCATCTTCTGT	3205
	TCTTTGAG**A**GGTGTTAA	3206
	TTAACACC**T**CTCAAAGA	3207

EXAMPLE 18
Human mismatch repair - MSH2

The human MSH2 gene is homologous to the bacterial *mutS* gene, which is involved in mismatch repair. Mutations in the MSH2 gene have been identified in a variety of cancers, including, for

example, ovarian tumors, colorectal cancer, endometrial cancer, uterine cancer. The attached table discloses the correcting oligonucleotide base sequences for the MSH2 oligonucleotides of the invention.

Table 25
MSH2 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non polyposis colorectal cancer Gln252Term CAG-TAG	TTTTCCACAAAAGACATTTATCAGGACCTCAACCGGTTGTTGA AAGGCAAAAAGGGAGAGCAGATGAATAGTGCTGTATTGCCAG AAATGGAGAATCAGGTACATGGATTATAAATGTGAA	3208
	TTCACATTTATAATCCATGTACCTGATTCTCCATTTCTGGCAAT ACAGCACTATTCATCTGCTCTCCCTTTTTGCCTTTCAACAACC GGTTGAGGTCCTGATAAATGTCTTTTGTGGAAAA	3209
	AGGGAGAGCAGATGAAT	3210
	ATTCATCTGCTCTCCCT	3211
Non polyposis colorectal cancer Gln288Term CAG-TAG	TCATCACTGTCTGCGGTAATCAAGTTTTTAGAACTCTTATCAG ATGATTCCAACTTTGGACAGTTTGAACTGACTACTTTTGACTT CAGCCAGTATATGAAATTGGATATTGCAGCAGTCA	3212
	TGACTGCTGCAATATCCAATTTCATATACTGGCTGAAGTCAAA AGTAGTCAGTTCAAACTGTCCAAAGTTGGAATCATCTGATAAG AGTTCTAAAAACTTGATTACCGCAGACAGTGATGA	3213
	ACTTTGGACAGTTTGAA	3214
	TTCAAACTGTCCAAAGT	3215
Non polyposis colorectal cancer Ala305Thr GCA-ACA	AACTTTGGACAGTTTGAACTGACTACTTTTGACTTCAGCCAGT ATATGAAATTGGATATTGCAGCAGTCAGAGCCCTTAACCTTTT TCAGGTAAAAAAAAAAAAAAAAAAAAAAAAAAAGG	3216
	CCTTTTTTTTTTTTTTTTTTTTTTTTTTTTACCTGAAAAAGGTTAAG GGCTCTGACTGCTGCAATATCCAATTTCATATACTGGCTGAAG TCAAAAGTAGTCAGTTCAAACTGTCCAAAGTT	3217
	TGGATATTGCAGCAGTC	3218
	GACTGCTGCAATATCCA	3219
Non polyposis colorectal cancer Gly322Asp GGC-GAC	AGCTTGCCATTCTTTCTATTTTATTTTTTGTTTACTAGGGTTCT GTTGAAGATACCACTGGCTCTCAGTCTCTGGCTGCCTTGCTG AATAAGTGTAAAACCCCTCAAGGACAAAGACTTGT	3220
	ACAAGTCTTTGTCCTTGAGGGGTTTTACACTTATTCAGCAAGG CAGCCAGAGACTGAGAGCCAGTGGTATCTTCAACAGAACCCT AGTAAACAAAAAATAAAATAGAAAGAATGGCAAGCT	3221
	TACCACTGGCTCTCAGT	3222
	ACTGAGAGCCAGTGGTA	3223
Non polyposis colorectal cancer Ser323Cys TCT-TGT	TTGCCATTCTTTCTATTTTATTTTTTGTTTACTAGGGTTCTGTTG AAGATACCACTGGCTCTCAGTCTCTGGCTGCCTTGCTGAATA AGTGTAAAACCCCTCAAGGACAAAGACTTGTTAA	3224
	TTAACAAGTCTTTGTCCTTGAGGGGTTTTACACTTATTCAGCA AGGCAGCCAGAGACTGAGAGCCAGTGGTATCTTCAACAGAAC CCTAGTAAACAAAAAATAAAATAGAAAGAATGGCAA	3225

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CACTGGCTCTCAGTCTC	3226
	GAGACTGAGAGCCAGTG	3227
Non polyposis colorectal cancer Arg383Term CGA-TGA	GTGGAAGCTTTTGTAGAAGATGCAGAATTGAGGCAGACTTTA CAAGAAGATTTACTTCGTCGATTCCCAGATCTTAACCGACTTG CCAAGAAGTTTCAAAGACAAGCAGCAAACTTACAAG	3228
	CTTGTAAGTTTGCTGCTTGTCTTTGAAACTTCTTGGCAAGTCG GTTAAGATCTGGGAATCGACGAAGTAAATCTTCTTGTAAAGTC TGCCTCAATTCTGCATCTTCTACAAAAGCTTCCAC	3229
	TACTTCGTCGATTCCCA	3230
	TGGGAATCGACGAAGTA	3231
Non polyposis colorectal cancer Gln397Term CAA-TAA	CAAGAAGATTTACTTCGTCGATTCCCAGATCTTAACCGACTTG CCAAGAAGTTTCAAAGACAAGCAGCAAACTTACAAGATTGTTA CCGACTCTATCAGGGTATAAATCAACTACCTAATG	3232
	CATTAGGTAGTTGATTTATACCCTGATAGAGTCGGTAACAATC TTGTAAGTTTGCTGCTTGTCTTTGAAACTTCTTGGCAAGTCGG TTAAGATCTGGGAATCGACGAAGTAAATCTTCTTG	3233
	TTCAAAGACAAGCAGCA	3234
	TGCTGCTTGTCTTTGAA	3235
Non polyposis colorectal cancer Arg406Term CGA-TGA	GATCTTAACCGACTTGCCAAGAAGTTTCAAAGACAAGCAGCA AACTTACAAGATTGTTACCGACTCTATCAGGGTATAAATCAAC TACCTAATGTTATACAGGCTCTGGAAAAACATGAAG	3236
	CTTCATGTTTTTCCAGAGCCTGTATAACATTAGGTAGTTGATTT ATACCCTGATAGAGTCGGTAACAATCTTGTAAGTTTGCTGCTT GTCTTTGAAACTTCTTGGCAAGTCGGTTAAGATC	3237
	ATTGTTACCGACTCTAT	3238
	ATAGAGTCGGTAACAAT	3239
Non polyposis colorectal cancer Gln419Term CAG-TAG	GCAAACTTACAAGATTGTTACCGACTCTATCAGGGTATAAATC AACTACCTAATGTTATACAGGCTCTGGAAAAACATGAAGGTAA CAAGTGATTTTGTTTTTTTGTTTTCCTTCAACTCA	3240
	TGAGTTGAAGGAAAACAAAAAAACAAAATCACTTGTTACCTTC ATGTTTTTCCAGAGCCTGTATAACATTAGGTAGTTGATTTATAC CCTGATAGAGTCGGTAACAATCTTGTAAGTTTGC	3241
	ATGTTATACAGGCTCTG	3242
	CAGAGCCTGTATAACAT	3243
Non polyposis colorectal cancer Gln429Term CAG-TAG	TATTCTGTAAAATGAGATCTTTTTATTTGTTTGTTTTACTACTTT CTTTTAGGAAAACACCAGAAATTATTGTTGGCAGTTTTTGTGA CTCCTCTTACTGATCTTCGTTCTGACTTCTCCA	3244
	TGGAGAAGTCAGAACGAAGATCAGTAAGAGGAGTCACAAAAA CTGCCAACAATAATTTCTGGTGTTTTCCTAAAAGAAAGTAGTA AAACAAACAAATAAAAAGATCTCATTTTACAGAATA	3245
	GAAAACACCAGAAATTA	3246
	TAATTTCTGGTGTTTTC	3247

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non polyposis colorectal cancer Leu458Term TTA-TGA	CTCCTCTTACTGATCTTCGTTCTGACTTCTCCAAGTTTCAGGA AATGATAGAAACAACTTTAGATATGGATCAGGTATGCAATATA CTTTTTAATTTAAGCAGTAGTTATTTTTAAAAAGC	3248
	GCTTTTTAAAAATAACTACTGCTTAAATTAAAAAGTATATTGCA TACCTGATCCATATCTAAAGTTGTTTCTATCATTTCCTGAAACT TGGAGAAGTCAGAACGAAGATCAGTAAGAGGAG	3249
	AACAACTTTAGATATGG	3250
	CCATATCTAAAGTTGTT	3251
Non polyposis colorectal cancer Gln518Term CAG-TAG	TTTCTTCTTGATTATCAAGGCTTGGACCCTGGCAAACAGATTA AACTGGATTCCAGTGCACAGTTTGGATATTACTTTCGTGTAAC CTGTAAGGAAGAAAAAGTCCTTCGTAACAATAAAA	3252
	TTTTATTGTTACGAAGGACTTTTTCTTCCTTACAGGTTACACGA AAGTAATATCCAAACTGTGCACTGGAATCCAGTTTAATCTGTT TGCCAGGGTCCAAGCCTTGATAATCAAGAAGAAA	3253
	CCAGTGCACAGTTTGGA	3254
	TCCAAACTGTGCACTGG	3255
Non polyposis colorectal cancer Arg524Pro CGT-CCT	GCTTGGACCCTGGCAAACAGATTAAACTGGATTCCAGTGCAC AGTTTGGATATTACTTTCGTGTAACCTGTAAGGAAGAAAAAGT CCTTCGTAACAATAAAAACTTTAGTACTGTAGATAT	3256
	ATATCTACAGTACTAAAGTTTTTATTGTTACGAAGGACTTTTTC TTCCTTACAGGTTACACGAAAGTAATATCCAAACTGTGCACTG GAATCCAGTTTAATCTGTTTGCCAGGGTCCAAGC	3257
	TTACTTTCGTGTAACCT	3258
	AGGTTACACGAAAGTAA	3259
Non polyposis colorectal cancer Glu562Val GAG-GTG	TTAATATTTTTAATAAAACTGTTATTTCGATTTGCAGCAAATTGA CTTCTTTAAATGAAGAGTATACCAAAAATAAAACAGAATATGAA GAAGCCCAGGATGCCATTGTTAAAGAAATTGT	3260
	ACAATTTCTTTAACAATGGCATCCTGGGCTTCTTCATATTCTGT TTTATTTTTGGTATACTCTTCATTTAAAGAAGTCAATTTGCTGC AAATCGAAATAACAGTTTTATTAAAAATATTAA	3261
	AAATGAAGAGTATACCA	3262
	TGGTATACTCTTCATTT	3263
Glioma Glu580Term GAA-TAA	AATGAAGAGTATACCAAAAATAAAACAGAATATGAAGAAGCCC AGGATGCCATTGTTAAAGAAATTGTCAATATTTCTTCAGGTAAA CTTAATAGAACTAATAATGTTCTGAATGTCACCT	3264
	AGGTGACATTCAGAACATTATTAGTTCTATTAAGTTTACCTGAA GAAATATTGACAATTTCTTTAACAATGGCATCCTGGGCTTCTT CATATTCTGTTTTATTTTTGGTATACTCTTCATT	3265
	TTGTTAAAGAAATTGTC	3266
	GACAATTTCTTTAACAA	3267
Non polyposis colorectal cancer Gln601Term CAG-TAG	TGTTTTTATTTTTATACAGGCTATGTAGAACCAATGCAGACACT CAATGATGTGTTAGCTCAGCTAGATGCTGTTGTCAGCTTTGCT CACGTGTCAAATGGAGCACCTGTTCCATATGTAC	3268

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GTACATATGGAACAGGTGCTCCATTTGACACGTGAGCAAAGC TGACAACAGCATCTAGCTGAGCTAACACATCATTGAGTGTCTG CATTGGTTCTACATAGCCTGTATAAAAATAAAAACA	3269
	TGTTAGCTCAGCTAGAT	3270
	ATCTAGCTGAGCTAACA	3271
Non polyposis colorectal cancer Tyr619Term TAT-TAG	AGCTCAGCTAGATGCTGTTGTCAGCTTTGCTCACGTGTCAAAT GGAGCACCTGTTCCATATGTACGACCAGCCATTTTGGAGAAA GGACAAGGAAGAATTATATTAAAAGCATCCAGGCAT	3272
	ATGCCTGGATGCTTTTAATATAATTCTTCCTTGTCCTTTCTCCA AAATGGCTGGTCGTACATATGGAACAGGTGCTCCATTTGACA CGTGAGCAAAGCTGACAACAGCATCTAGCTGAGCT	3273
	GTTCCATATGTACGACC	3274
	GGTCGTACATATGGAAC	3275
Non polyposis colorectal cancer Arg621Term CGA-TGA	CAGCTAGATGCTGTTGTCAGCTTTGCTCACGTGTCAAATGGA GCACCTGTTCCATATGTACGACCAGCCATTTTGGAGAAAGGA CAAGGAAGAATTATATTAAAAGCATCCAGGCATGCTT	3276
	AAGCATGCCTGGATGCTTTTAATATAATTCTTCCTTGTCCTTTC TCCAAAATGGCTGGTCGTACATATGGAACAGGTGCTCCATTT GACACGTGAGCAAAGCTGACAACAGCATCTAGCTG	3277
	CATATGTACGACCAGCC	3278
	GGCTGGTCGTACATATG	3279
Non polyposis colorectal cancer Pro622Leu CCA-CTA	TAGATGCTGTTGTCAGCTTTGCTCACGTGTCAAATGGAGCAC CTGTTCCATATGTACGACCAGCCATTTTGGAGAAAGGACAAG GAAGAATTATATTAAAAGCATCCAGGCATGCTTGTGT	3280
	ACACAAGCATGCCTGGATGCTTTTAATATAATTCTTCCTTGTC CTTTCTCCAAAATGGCTGGTCGTACATATGGAACAGGTGCTC CATTTGACACGTGAGCAAAGCTGACAACAGCATCTA	3281
	TGTACGACCAGCCATTT	3282
	AAATGGCTGGTCGTACA	3283
Non polyposis colorectal cancer Ala636Pro GCA-CCA	CCTGTTCCATATGTACGACCAGCCATTTTGGAGAAAGGACAA GGAAGAATTATATTAAAAGCATCCAGGCATGCTTGTGTTGAAG TTCAAGATGAAATTGCATTTATTCCTAATGACGTAT	3284
	ATACGTCATTAGGAATAAATGCAATTTCATCTTGAACTTCAACA CAAGCATGCCTGGATGCTTTTAATATAATTCTTCCTTGTCCTTT CTCCAAAATGGCTGGTCGTACATATGGAACAGG	3285
	TATTAAAAGCATCCAGG	3286
	CCTGGATGCTTTTAATA	3287
Non polyposis colorectal cancer His639Arg CAT-CGT	ATGTACGACCAGCCATTTTGGAGAAAGGACAAGGAAGAATTA TATTAAAAGCATCCAGGCATGCTTGTGTTGAAGTTCAAGATGA AATTGCATTTATTCCTAATGACGTATACTTTGAAAA	3288
	TTTTCAAAGTATACGTCATTAGGAATAAATGCAATTTCATCTTG AACTTCAACACAAGCATGCCTGGATGCTTTTAATATAATTCTTC CTTGTCCTTTCTCCAAAATGGCTGGTCGTACAT	3289
	ATCCAGGCATGCTTGTG	3290

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CACAAGCATGCCTGGAT	3291
Non polyposis colorectal cancer His639Tyr CAT-TAT	TATGTACGACCAGCCATTTTGGAGAAAGGACAAGGAAGAATT ATATTAAAAGCATCCAGGCATGCTTGTGTTGAAGTTCAAGATG AAATTGCATTTATTCCTAATGACGTATACTTTGAAA	3292
	TTTCAAAGTATACGTCATTAGGAATAAATGCAATTTCATCTTGA ACTTCAACACAAGCATGCCTGGATGCTTTTAATATAATTCTTC CTTGTCCTTTCTCCAAAATGGCTGGTCGTACATA	3293
	CATCCAGGCATGCTTGT	3294
	ACAAGCATGCCTGGATG	3295
Non polyposis colorectal cancer Glu647Lys GAA-AAA	AAAGGACAAGGAAGAATTATATTAAAAGCATCCAGGCATGCTT GTGTTGAAGTTCAAGATGAAATTGCATTTATTCCTAATGACGT ATACTTTGAAAAAGATAAACAGATGTTCCACATCA	3296
	TGATGTGGAACATCTGTTTATCTTTTTCAAAGTATACGTCATTA GGAATAAATGCAATTTCATCTTGAACTTCAACACAAGCATGCC TGGATGCTTTTAATATAATTCTTCCTTGTCCTTT	3297
	TTCAAGATGAAATTGCA	3298
	TGCAATTTCATCTTGAA	3299
Non polyposis colorectal cancer Tyr656Term TAC-TAG	ATCCAGGCATGCTTGTGTTGAAGTTCAAGATGAAATTGCATTT ATTCCTAATGACGTATACTTTGAAAAAGATAAACAGATGTTCCA CATCATTACTGGTAAAAAACCTGGTTTTTGGGCT	3300
	AGCCCAAAAACCAGGTTTTTTACCAGTAATGATGTGGAACATC TGTTTATCTTTTTCAAAGTATACGTCATTAGGAATAAATGCAAT TTCATCTTGAACTTCAACACAAGCATGCCTGGAT	3301
	GACGTATACTTTGAAAA	3302
	TTTTCAAAGTATACGTC	3303
Non polyposis colorectal cancer Gly674Asp GGT-GAT	GAAAGAAGTTTAAAATCTTGCTTTCTGATATAATTTGTTTTGTA GGCCCCAATATGGGAGGTAAATCAACATATATTCGACAAACT GGGGTGATAGTACTCATGGCCCAAATTGGGTGTTT	3304
	AAACACCCAATTTGGGCCATGAGTACTATCACCCCAGTTTGTC GAATATATGTTGATTTACCTCCCATATTGGGGCCTACAAAACA AATTATATCAGAAAGCAAGATTTTAAACTTCTTTC	3305
	TATGGGAGGTAAATCAA	3306
	TTGATTTACCTCCCATA	3307
Non polyposis colorectal cancer Arg680Term CGA-TGA	TTGCTTTCTGATATAATTTGTTTTGTAGGCCCCAATATGGGAG GTAAATCAACATATATTCGACAAACTGGGGTGATAGTACTCAT GGCCCAAATTGGGTGTTTTGTGCCATGTGAGTCAG	3308
	CTGACTCACATGGCACAAAACACCCAATTTGGGCCATGAGTA CTATCACCCCAGTTTGTCGAATATATGTTGATTTACCTCCCAT ATTGGGGCCTACAAAACAAATTATATCAGAAAGCAA	3309
	CATATATTCGACAAACT	3310
	AGTTTGTCGAATATATG	3311

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non polyposis colorectal cancer Gly692Arg GGG-CGG	ATGGGAGGTAAATCAACATATATTCGACAAACTGGGGTGATA GTACTCATGGCCCAAATTGGGTGTTTTGTGCCATGTGAGTCA GCAGAAGTGTCCATTGTGGACTGCATCTTAGCCCGAG	3312
	CTCGGGCTAAGATGCAGTCCACAATGGACACTTCTGCTGACT CACATGGCACAAAACACCCAATTTGGGCCATGAGTACTATCA CCCCAGTTTGTCGAATATATGTTGATTTACCTCCCAT	3313
	CCCAAATTGGGTGTTTT	3314
	AAAACACCCAATTTGGG	3315
Non polyposis colorectal cancer Cys697Arg TGT-CGT	ACATATATTCGACAAACTGGGGTGATAGTACTCATGGCCCAAA TTGGGTGTTTTGTGCCATGTGAGTCAGCAGAAGTGTCCATTG TGGACTGCATCTTAGCCCGAGTAGGGGCTGGTGACA	3316
	TGTCACCAGCCCCTACTCGGGCTAAGATGCAGTCCACAATGG ACACTTCTGCTGACTCACATGGCACAAAACACCCAATTTGGG CCATGAGTACTATCACCCCAGTTTGTCGAATATATGT	3317
	TTGTGCCATGTGAGTCA	3318
	TGACTCACATGGCACAA	3319
Non polyposis colorectal cancer Cys697Phe TGT-TTT	CATATATTCGACAAACTGGGGTGATAGTACTCATGGCCCAAAT TGGGTGTTTTGTGCCATGTGAGTCAGCAGAAGTGTCCATTGT GGACTGCATCTTAGCCCGAGTAGGGGCTGGTGACAG	3320
	CTGTCACCAGCCCCTACTCGGGCTAAGATGCAGTCCACAATG GACACTTCTGCTGACTCACATGGCACAAAACACCCAATTTGG GCCATGAGTACTATCACCCCAGTTTGTCGAATATATG	3321
	TGTGCCATGTGAGTCAG	3322
	CTGACTCACATGGCACA	3323
Non polyposis colorectal cancer Gln718Term CAA-TAA	GAGTCAGCAGAAGTGTCCATTGTGGACTGCATCTTAGCCCGA GTAGGGGCTGGTGACAGTCAATTGAAAGGAGTCTCCACGTTC ATGGCTGAAATGTTGGAAACTGCTTCTATCCTCAGGT	3324
	ACCTGAGGATAGAAGCAGTTTCCAACATTTCAGCCATGAACG TGGAGACTCCTTTCAATTGACTGTCACCAGCCCCTACTCGGG CTAAGATGCAGTCCACAATGGACACTTCTGCTGACTC	3325
	GTGACAGTCAATTGAAA	3326
	TTTCAATTGACTGTCAC	3327
Non polyposis colorectal cancer Leu811Term TTA-TGA	CCAATCAGATACCAACTGTTAATAATCTACATGTCACAGCACT CACCACTGAAGAGACCTTAACTATGCTTTATCAGGTGAAGAAA GGTATGTACTATTGGAGTACTCTAAATTCAGAACT	3328
	AGTTCTGAATTTAGAGTACTCCAATAGTACATACCTTTCTTCAC CTGATAAAGCATAGTTAAGGTCTCTTCAGTGGTGAGTGCTGT GACATGTAGATTATTAACAGTTGGTATCTGATTGG	3329
	AGAGACCTTAACTATGC	3330
	GCATAGTTAAGGTCTCT	3331
Non polyposis colorectal cancer Ala834Thr GCT-ACT	TTCCCCAAATTTCTTATAGGTGTCTGTGATCAAAGTTTTGGGA TTCATGTTGCAGAGCTTGCTAATTTCCCTAAGCATGTAATAGA GTGTGCTAAACAGAAAGCCCTGGAACTTGAGGAGT	3332

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACTCCTCAAGTTCCAGGGCTTTCTGTTTAGCACACTCTATTAC ATGCTTAGGGAAATTAGCAAGCTCTGCAACATGAATCCCAAAA CTTTGATCACAGACACCTATAAGAAATTTGGGGAA	3333
	CAGAGCTTGCTAATTTC	3334
	GAAATTAGCAAGCTCTG	3335
Non polyposis colorectal cancer Gln861Term CAA-TAA	ATAGAGTGTGCTAAACAGAAAGCCCTGGAACTTGAGGAGTTT CAGTATATTGGAGAATCGCAAGGATATGATATCATGGAACCAG CAGCAAAGAAGTGCTATCTGGAAAGAGAGGTTTGTC	3336
	GACAAACCTCTCTTTCCAGATAGCACTTCTTTGCTGCTGGTTC CATGATATCATATCCTTGCGATTCTCCAATATACTGAAACTCCT CAAGTTCCAGGGCTTTCTGTTTAGCACACTCTAT	3337
	GAGAATCGCAAGGATAT	3338
	ATATCCTTGCGATTCTC	3339
Non polyposis colorectal cancer Thr905Arg ACA-AGA	AGGAGTTCCTGTCCAAGGTGAAACAAATGCCCTTTACTGAAAT GTCAGAAGAAAACATCACAATAAAGTTAAAACAGCTAAAAGCT GAAGTAATAGCAAAGAATAATAGCTTTGTAAATGA	3340
	TCATTTACAAAGCTATTATTCTTTGCTATTACTTCAGCTTTTAG CTGTTTTAACTTTATTGTGATGTTTTCTTCTGACATTTCAGTAA AGGGCATTTGTTTCACCTTGGACAGGAACTCCT	3341
	AAACATCACAATAAAGT	3342
	ACTTTATTGTGATGTTT	3343

EXAMPLE 19
Human mismatch repair - MSH6

The human MSH6 gene is homologous to the bacterial *mutS* gene, which is involved in mismatch repair. Mutations in the MSH6 gene have been identified in a variety of cancers, including particularly hereditary nonpolyposis colorectal cancer. The attached table discloses the correcting oligonucleotide base sequences for the MSH6 oligonucleotides of the invention.

Table 26
MSH6 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non-polyposis colorectal cancer Ser144Ile AGC-ATC	GGAAATCAGTCCGTGTTCATGTACAGTTTTTTGATGACAGCCC AACAAGGGGCTGGGTTAGCAAAAGGCTTTTAAAGCCATATAC AGGTAAGAGTCACTACTGCCATGTGTGTGTGTTTGT	3344

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACAAACACACACACATGGCAGTAGTGACTCTTACCTGTATATG GCTTTAAAAGCCTTTTGCTAACCCAGCCCCTTGTTGGGCTGT CATCAAAAAACTGTACATGAACACGGACTGATTTCC	3345
	CTGGGTTAGCAAAAGGC	3346
	GCCTTTTGCTAACCCAG	3347
Endometrial cancer Ser156Term TCA-TGA	CGTGAGCCTCTGCACCCGGCCCTTATTGTTTATAAATACATTT CTTTCTAGGTTCAAAATCAAAGGAAGCCCAGAAGGGAGGTCA TTTTTACAGTGCAAAGCCTGAAATACTGAGAGCAAT	3348
	ATTGCTCTCAGTATTTCAGGCTTTGCACTGTAAAAATGACCTC CCTTCTGGGCTTCCTTTGATTTTGAACCTAGAAAGAAATGTAT TTATAAACAATAAGGGCCGGGTGCAGAGGCTCACG	3349
	TTCAAAATCAAAGGAAG	3350
	CTTCCTTTGATTTTGAA	3351
Early onset colorectal cancer Tyr214Term TAC-TAG	TTCCAAATTTTGATTTGTTTTTAAATACTCTTTCCTTGCCTGGC AGGTAGGCACAACTTACGTAACAGATAAGAGTGAAGAAGATA ATGAAATTGAGAGTGAAGAGGAAGTACAGCCTAAG	3352
	CTTAGGCTGTACTTCCTCTTCACTCTCAATTTCATTATCTTCTT CACTCTTATCTGTTACGTAAGTTGTGCCTACCTGCCAGGCAA GGAAAGAGTATTTAAAAACAAATCAAAATTTGGAA	3353
	ACAACTTACGTAACAGA	3354
	TCTGTTACGTAAGTTGT	3355
Endometrial cancer Arg248Term CGA-TGA	GAAGAGGAAGTACAGCCTAAGACACAAGGATCTAGGCGAAGT AGCCGCCAAATAAAAAAACGAAGGGTCATATCAGATTCTGAG AGTGACATTGGTGGCTCTGATGTGGAATTTAAGCCAG	3356
	CTGGCTTAAATTCCACATCAGAGCCACCAATGTCACTCTCAGA ATCTGATATGACCCTTCGTTTTTTTATTTGGCGGCTACTTCGC CTAGATCCTTGTGTCTTAGGCTGTACTTCCTCTTC	3357
	TAAAAAAACGAAGGGTC	3358
	GACCCTTCGTTTTTTTA	3359
Colorectal cancer Ser285Ile AGT-ATT	TTAAGCCAGACACTAAGGAGGAAGGAAGCAGTGATGAAATAA GCAGTGGAGTGGGGGATAGTGAGAGTGAAGGCCTGAACAGC CCTGTCAAAGTTGCTCGAAAGCGGAAGAGAATGGTGAC	3360
	GTCACCATTCTCTTCCGCTTTCGAGCAACTTTGACAGGGCTG TTCAGGCCTTCACTCTCACTATCCCCCACTCCACTGCTTATTT CATCACTGCTTCCTTCCTCCTTAGTGTCTGGCTTAA	3361
	GGGGGATAGTGAGAGTG	3362
	CACTCTCACTATCCCCC	3363
Colorectal cancer Gly566Arg GGA-AGA	GAGGAAGATTCTTCTGGCCATACTCGTGCATATGGTGTGTGC TTTGTTGATACTTCACTGGGAAAGTTTTTCATAGGTCAGTTTTC AGATGATCGCCATTGTTCGAGATTTAGGACTCTAG	3364

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTAGAGTCCTAAATCTCGAACAATGGCGATCATCTGAAAACTG ACCTATGAAAAACTTTCCCAGTGAAGTATCAACAAAGCACACA CCATATGCACGAGTATGGCCAGAAGAATCTTCCTC	3365
	CTTCACTGGGAAAGTTT	3366
	AAACTTTCCCAGTGAAG	3367
Non-polyposis colorectal cancer Gln698Glu CAG-GAG	GAATTGGCCCTCTCTGCTCTAGGTGGTTGTGTCTTCTACCTC AAAAAATGCCTTATTGATCAGGAGCTTTTATCAATGGCTAATTT TGAAGAATATATTCCCTTGGATTCTGACACAGTCA	3368
	TGACTGTGTCAGAATCCAAGGGAATATATTCTTCAAAATTAGC CATTGATAAAAGCTCCTGATCAATAAGGCATTTTTTGAGGTAG AAGACACAACCACCTAGAGCAGAGAGGGCCAATTC	3369
	TTATTGATCAGGAGCTT	3370
	AAGCTCCTGATCAATAA	3371
Endometrial cancer Gln731Term CAA-TAA	CCCTTGGATTCTGACACAGTCAGCACTACAAGATCTGGTGCT ATCTTCACCAAAGCCTATCAACGAATGGTGCTAGATGCAGTG ACATTAAACAACTTGGAGATTTTTCTGAATGGAACAA	3372
	TTGTTCCATTCAGAAAAATCTCCAAGTTGTTTAATGTCACTGCA TCTAGCACCATTCGTTGATAGGCTTTGGTGAAGATAGCACCA GATCTTGTAGTGCTGACTGTGTCAGAATCCAAGGG	3373
	AAGCCTATCAACGAATG	3374
	CATTCGTTGATAGGCTT	3375
Colorectal cancer Val800Leu GTT-CTT	GCCCCACTCTGTAACCATTATGCTATTAATGATCGTCTAGATG CCATAGAAGACCTCATGGTTGTGCCTGACAAAATCTCCGAAG TTGTAGAGCTTCTAAAGAAGCTTCCAGATCTTGAGA	3376
	TCTCAAGATCTGGAAGCTTCTTTAGAAGCTCTACAACTTCGGA GATTTTGTCAGGCACAACCATGAGGTCTTCTATGGCATCTAGA CGATCATTAATAGCATAATGGTTACAGAGTGGGGC	3377
	ACCTCATGGTTGTGCCT	3378
	AGGCACAACCATGAGGT	3379
Colorectal cancer Asp803Gly GAC-GGC	GTAACCATTATGCTATTAATGATCGTCTAGATGCCATAGAAGA CCTCATGGTTGTGCCTGACAAAATCTCCGAAGTTGTAGAGCT TCTAAAGAAGCTTCCAGATCTTGAGAGGCTACTCAG	3380
	CTGAGTAGCCTCTCAAGATCTGGAAGCTTCTTTAGAAGCTCTA CAACTTCGGAGATTTTGTTCAGGCACAACCATGAGGTCTTCTAT GGCATCTAGACGATCATTAATAGCATAATGGTTAC	3381
	TGTGCCTGACAAAATCT	3382
	AGATTTTGTTCAGGCACA	3383
Non-polyposis colorectal cancer Tyr850Cys TAC-TGC	CTCCCCTGAAGAGTCAGAACCACCCAGACAGCAGGGCTATAA TGTATGAAGAAACTACATACAGCAAGAAGAAGATTATTGATTT TCTTTCTGCTCTGGAAGGATTCAAAGTAATGTGTAA	3384

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTACACATTACTTTGAATCCTTCCAGAGCAGAAAGAAAATCAA TAATCTTCTTCTTGCTGTATGTAGTTTCTTCATACATTATAGCC CTGCTGTCTGGGTGGTTCTGACTCTTCAGGGGAG	3385
	AACTACATACAGCAAGA	3386
	TCTTGCTGTATGTAGTT	3387
Colorectal cancer Pro1087Thr CCC-ACC	TATAGTCGAGGGGGTGATGGTCCTATGTGTCGCCCAGTAATT CTGTTGCCGGAAGATACCCCCCCCTTCTTAGAGCTTAAAGGA TCACGCCATCCTTGCATTACGAAGACTTTTTTTGGAG	3388
	CTCCAAAAAAAAGTCTTCGTAATGCAAGGATGGCGTGATCCTTT AAGCTCTAAGAAGGGGGGGGGTATCTTCCGGCAACAGAATTAC TGGGCGACACATAGGACCATCACCCCCTCGACTATA	3389
	AAGATACCCCCCCCTTC	3390
	GAAGGGGGGGGGTATCTT	3391
Non-polyposis colorectal cancer Gln1258Term CAA-TAA	ACTATAAAATGTCGTACATTATTTTCAACTCACTACCATTCATT AGTAGAAGATTATTCTCAAAATGTTGCTGTGCGCCTAGGACAT ATGGTATGTGCAAATTGTTTTTTTCCACAAATTC	3392
	GAATTTGTGGAAAAAAACAATTTGCACATACCATATGTCCTAG GCGCACAGCAACATTTTGAGAATAATCTTCTACTAATGAATGG TAGTGAGTTGAAAATAATGTACGACATTTTATAGT	3393
	ATTATTCTCAAAATGTT	3394
	AACATTTTGAGAATAAT	3395

EXAMPLE 20
Hyperlipidemia - APOE

Hyperlipidemia is the abnormal elevation of plasma cholesterol and/or triglyceride levels and it is one of the most common diseases. The human apolipoprotein E protein is involved in the transport of endogenous lipids and appears to be crucial for both the direct removal of cholesterol-rich LDL from plasma and conversion of IDL particles to LDL particles. Individuals who either lack apolipoprotein E or who are homozygous for particular alleles of apoE may have have a condition known as dysbetalipoproteinemia, which is characterized by elevated plasma cholesterol and triglyceride levels and an increased risk for atherosclerosis.

In a comprehensive review of apoE variants, de Knijff et al., *Hum. Mutat.* 4:178-194 (1994) found that 30 variants had been characterized, including the most common variant, apoE3. To that time, 14 apoE variants had been found to be associated with familial dysbetalipoproteinemia. The

attached table discloses the correcting oligonucleotide base sequences for the APOE oligonucleotides of the invention.

Table 27
APOE Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Apolipoprotein E Glu13Lys cGAG-AAG	TTGTTCCACACAGGATGCCAGGCCAAGGTGGAGCAAGCGGT GGAGACAGAGCCGGAGCCC**G**AGCTGCGCCAGCAGACCGAG TGGCAGAGCGGCCAGCGCTGGGAACTGGCACTGGGTCGCT	3396
	AGCGACCCAGTGCCAGTTCCCAGCGCTGGCCGCTCTGCCAC TCGGTCTGCTGGCGCAGCT**C**GGGCTCCGGCTCTGTCTCCAC CGCTTGCTCCACCTTGGCCTGGCATCCTGTGTGGAACAA	3397
	CGGAGCCC**G**AGCTGCGC	3398
	GCGCAGCT**C**GGGCTCCG	3399
Apolipoprotein E Trp20Term TGGc-TGA	CAAGGTGGAGCAAGCGGTGGAGACAGAGCCGGAGCCCGAG CTGCGCCAGCAGACCGAGTG**G**CAGAGCGGCCAGCGCTGGG AACTGGCACTGGGTCGCTTTTGGGATTACCTGCGCTGGGTG	3400
	CACCCAGCGCAGGTAATCCCAAAAGCGACCCAGTGCCAGTT CCCAGCGCTGGCCGCTCTG**C**CACTCGGTCTGCTGGCGCAGC TCGGGCTCCGGCTCTGTCTCCACCGCTTGCTCCACCTTG	3401
	ACCGAGTG**G**CAGAGCGG	3402
	CCGCTCTG**C**CACTCGGT	3403
Apolipoprotein E Leu28Pro CTG-CCG	CAGAGCCGGAGCCCGAGCTGCGCCAGCAGACCGAGTGGCA GAGCGGCCAGCGCTGGGAAC**T**GGCACTGGGTCGCTTTTGGG ATTACCTGCGCTGGGTGCAGACACTGTCTGAGCAGGTGCA	3404
	TGCACCTGCTCAGACAGTGTCTGCACCCAGCGCAGGTAATCC CAAAAGCGACCCAGTGCC**A**GTTCCCAGCGCTGGCCGCTCTG CCACTCGGTCTGCTGGCGCAGCTCGGGCTCCGGCTCTG	3405
	CTGGGAAC**T**GGCACTGG	3406
	CCAGTGCC**A**GTTCCCAG	3407
Apolipoprotein E Cys112Arg gTGC-CGC	CGGCTGTCCAAGGAGCTGCAGGCGGCGCAGGCCCGGCTGG GCGCGGACATGGAGGACGTG**T**GCGGCCGCCTGGTGCAGTA CCGCGGCGAGGTGCAGGCCATGCTCGGCCAGAGCACCGAG G	3408
	CCTCGGTGCTCTGGCCGAGCATGGCCTGCACCTCGCCGCGG TACTGCACCAGGCGGCCGC**A**CACGTCCTCCATGTCCGCGCC CAGCCGGGCCTGCGCCGCCTGCAGCTCCTTGGACAGCCG	3409
	AGGACGTG**T**GCGGCCGC	3410
	GCGGCCGC**A**CACGTCCT	3411
Apolipoprotein E Gly127Asp GGC-GAC	ACATGGAGGACGTGTGCGGCCGCCTGGTGCAGTACCGCGG CGAGGTGCAGGCCATGCTCG**G**CCAGAGCACCGAGGAGCTG CGGGTGCGCCTCGCCTCCCACCTGCGCAAGCTGCGTAAGCG	3412

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CGCTTACGCAGCTTGCGCAGGTGGGAGGCGAGGCGCACCC GCAGCTCCTCGGTGCTCTGG**C**CGAGCATGGCCTGCACCTCG CCGCGGTACTGCACCAGGCGGCCGCACACGTCCTCCATGT	3413
	CATGCTCG**G**CCAGAGCA	3414
	TGCTCTGG**C**CGAGCATG	3415
Apolipoprotein E Arg136Cys gCGC-TGC	GTGCAGTACCGCGGCGAGGTGCAGGCCATGCTCGGCCAGA GCACCGAGGAGCTGCGGGTG**C**GCCTCGCCTCCCACCTGCG CAAGCTGCGTAAGCGGCTCCTCCGCGATGCCGATGACCTGC	3416
	GCAGGTCATCGGCATCGCGGAGGAGCCGCTTACGCAGCTTG CGCAGGTGGGAGGCGAGGC**G**CACCCGCAGCTCCTCGGTGC TCTGGCCGAGCATGGCCTGCACCTCGCCGCGGTACTGCAC	3417
	TGCGGGTG**C**GCCTCGCC	3418
	GGCGAGGC**G**CACCCGCA	3419
Apolipoprotein E Arg136His CGC-CAC	TGCAGTACCGCGGCGAGGTGCAGGCCATGCTCGGCCAGAG CACCGAGGAGCTGCGGGTGC**G**CCTCGCCTCCCACCTGCGC AAGCTGCGTAAGCGGCTCCTCCGCGATGCCGATGACCTGCA	3420
	TGCAGGTCATCGGCATCGCGGAGGAGCCGCTTACGCAGCTT GCGCAGGTGGGAGGCGAGG**C**GCACCCGCAGCTCCTCGGTG CTCTGGCCGAGCATGGCCTGCACCTCGCCGCGGTACTGCA	3421
	GCGGGTGC**G**CCTCGCCT	3422
	AGGCGAGG**C**GCACCCGC	3423
Apolipoprotein E Arg136Ser gCGC-AGC	GTGCAGTACCGCGGCGAGGTGCAGGCCATGCTCGGCCAGA GCACCGAGGAGCTGCGGGTG**C**GCCTCGCCTCCCACCTGCG CAAGCTGCGTAAGCGGCTCCTCCGCGATGCCGATGACCTGC	3424
	GCAGGTCATCGGCATCGCGGAGGAGCCGCTTACGCAGCTTG CGCAGGTGGGAGGCGAGGC**G**CACCCGCAGCTCCTCGGTGC TCTGGCCGAGCATGGCCTGCACCTCGCCGCGGTACTGCAC	3425
	TGCGGGTG**C**GCCTCGCC	3426
	GGCGAGGC**G**CACCCGCA	3427
Apolipoprotein E Arg142Cys gCGC-TGC	GTGCAGGCCATGCTCGGCCAGAGCACCGAGGAGCTGCGGG TGCGCCTCGCCTCCCACCTG**C**GCAAGCTGCGTAAGCGGCTC CTCCGCGATGCCGATGACCTGCAGAAGCGCCTGGCAGTGT	3428
	ACACTGCCAGGCGCTTCTGCAGGTCATCGGCATCGCGGAGG AGCCGCTTACGCAGCTTGC**G**CAGGTGGGAGGCGAGGCGCA CCCGCAGCTCCTCGGTGCTCTGGCCGAGCATGGCCTGCAC	3429
	CCCACCTG**C**GCAAGCTG	3430
	CAGCTTGC**G**CAGGTGGG	3431
Apolipoprotein E Arg142Leu CGC-CTC	TGCAGGCCATGCTCGGCCAGAGCACCGAGGAGCTGCGGGT GCGCCTCGCCTCCCACCTGC**G**CAAGCTGCGTAAGCGGCTCC TCCGCGATGCCGATGACCTGCAGAAGCGCCTGGCAGTGTA	3432
	TACACTGCCAGGCGCTTCTGCAGGTCATCGGCATCGCGGAG GAGCCGCTTACGCAGCTTG**C**GCAGGTGGGAGGCGAGGCGC ACCCGCAGCTCCTCGGTGCTCTGGCCGAGCATGGCCTGCA	3433
	CCACCTGC**G**CAAGCTGC	3434

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCAGCTTGCGCAGGTGG	3435
Apolipoprotein E Arg145Cys gCGT-TGT	ATGCTCGGCCAGAGCACCGAGGAGCTGCGGGTGCGCCTCG CCTCCCACCTGCGCAAGCTGCGTAAGCGGCTCCTCCGCGAT GCCGATGACCTGCAGAAGCGCCTGGCAGTGTACCAGGCCG	3436
	CGGCCTGGTACACTGCCAGGCGCTTCTGCAGGTCATCGGCA TCGCGGAGGAGCCGCTTACGCAGCTTGCGCAGGTGGGAGG CGAGGCGCACCCGCAGCTCCTCGGTGCTCTGGCCGAGCAT	3437
	GCAAGCTGCGTAAGCGG	3438
	CCGCTTACGCAGCTTGC	3439
Apolipoprotein E Arg145Pro CGT-CCT	TGCTCGGCCAGAGCACCGAGGAGCTGCGGGTGCGCCTCGC CTCCCACCTGCGCAAGCTGCGTAAGCGGCTCCTCCGCGATG CCGATGACCTGCAGAAGCGCCTGGCAGTGTACCAGGCCGG	3440
	CCGGCCTGGTACACTGCCAGGCGCTTCTGCAGGTCATCGGC ATCGCGGAGGAGCCGCTTACGCAGCTTGCGCAGGTGGGAG GCGAGGCGCACCCGCAGCTCCTCGGTGCTCTGGCCGAGCA	3441
	CAAGCTGCGTAAGCGGC	3442
	GCCGCTTACGCAGCTTG	3443
Apolipoprotein E Lys146Gln tAAG-CAG	CTCGGCCAGAGCACCGAGGAGCTGCGGGTGCGCCTCGCCT CCCACCTGCGCAAGCTGCGTAAGCGGCTCCTCCGCGATGCC GATGACCTGCAGAAGCGCCTGGCAGTGTACCAGGCCGGGG	3444
	CCCCGGCCTGGTACACTGCCAGGCGCTTCTGCAGGTCATCG GCATCGCGGAGGAGCCGCTTACGCAGCTTGCGCAGGTGGGA GGCGAGGCGCACCCGCAGCTCCTCGGTGCTCTGGCCGAG	3445
	AGCTGCGTAAGCGGCTC	3446
	GAGCCGCTTACGCAGCT	3447
Apolipoprotein E Lys146Glu tAAG-GAG	CTCGGCCAGAGCACCGAGGAGCTGCGGGTGCGCCTCGCCT CCCACCTGCGCAAGCTGCGTAAGCGGCTCCTCCGCGATGCC GATGACCTGCAGAAGCGCCTGGCAGTGTACCAGGCCGGGG	3448
	CCCCGGCCTGGTACACTGCCAGGCGCTTCTGCAGGTCATCG GCATCGCGGAGGAGCCGCTTACGCAGCTTGCGCAGGTGGGA GGCGAGGCGCACCCGCAGCTCCTCGGTGCTCTGGCCGAG	3449
	AGCTGCGTAAGCGGCTC	3450
	GAGCCGCTTACGCAGCT	3451
Apolipoprotein E Arg158Cys gCGC-TGC	GCCTCCCACCTGCGCAAGCTGCGTAAGCGGCTCCTCCGCGA TGCCGATGACCTGCAGAAGCGCCTGGCAGTGTACCAGGCCG GGGCCCGCGAGGGCGCCGAGCGCGGCCTCAGCGCCATCC	3452
	GGATGGCGCTGAGGCCGCGCTCGGCGCCCTCGCGGGCCCC GGCCTGGTACACTGCCAGGCGCTTCTGCAGGTCATCGGCAT CGCGGAGGAGCCGCTTACGCAGCTTGCGCAGGTGGGAGGC	3453
	TGCAGAAGCGCCTGGCA	3454
	TGCCAGGCGCTTCTGCA	3455

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Apolipoprotein E Gln187Glu aCAG-GAG	CGCGAGGGCGCCGAGCGCGGCCTCAGCGCCATCCGCGAGC GCCTGGGGCCCCTGGTGGAA**C**AGGGCCGCGTGCGGGCCGC CACTGTGGGCTCCCTGGCCGGCCAGCCGCTACAGGAGCGG G	3456
	CCCGCTCCTGTAGCGGCTGGCCGGCCAGGGAGCCCACAGT GGCGGCCCGCACGCGGCCCT**G**TTCCACCAGGGGCCCCAGG CGCTCGCGGATGGCGCTGAGGCCGCGCTCGGCGCCCTCGC G	3457
	TGGTGGAA**C**AGGGCCGC	3458
	GCGGCCCT**G**TTCCACCA	3459
Apolipoprotein E Trp210Term TGG-TAG	TGCGGGCCGCCACTGTGGGCTCCCTGGCCGGCCAGCCGCT ACAGGAGCGGGCCCAGGCCT**G**GGGCGAGCGGCTGCGCGC GCGGATGGAGGAGATGGGCAGCCGGACCCGCGACCGCCTG GA	3460
	TCCAGGCGGTCGCGGGTCCGGCTGCCCATCTCCTCCATCCG CGCGCGCAGCCGCTCGCCC**C**AGGCCTGGGCCCGCTCCTGT AGCGGCTGGCCGGCCAGGGAGCCCACAGTGGCGGCCCGCA	3461
	CCAGGCCT**G**GGGCGAGC	3462
	GCTCGCCC**C**AGGCCTGG	3463
Apolipoprotein E Arg228Cys cCGC-TGC	CAGGCCTGGGGCGAGCGGCTGCGCGCGCGGATGGAGGAGA TGGGCAGCCGGACCCGCGAC**C**GCCTGGACGAGGTGAAGGA GCAGGTGGCGGAGGTGCGCGCCAAGCTGGAGGAGCAGGCC C	3464
	GGGCCTGCTCCTCCAGCTTGGCGCGCACCTCCGCCACCTGC TCCTTCACCTCGTCCAGGC**G**GTCGCGGGTCCGGCTGCCCAT CTCCTCCATCCGCGCGCGCAGCCGCTCGCCCCAGGCCTG	3465
	CCCGCGAC**C**GCCTGGAC	3466
	GTCCAGGC**G**GTCGCGGG	3467
Apolipoprotein E Glu244Lys gGAG-AAG	CGGACCCGCGACCGCCTGGACGAGGTGAAGGAGCAGGTGG CGGAGGTGCGCGCCAAGCTG**G**AGGAGCAGGCCCAGCAGAT ACGCCTGCAGGCCGAGGCCTTCCAGGCCCGCCTCAAGAGCT	3468
	AGCTCTTGAGGCGGGCCTGGAAGGCCTCGGCCTGCAGGCGT ATCTGCTGGGCCTGCTCCT**C**CAGCTTGGCGCGCACCTCCGC CACCTGCTCCTTCACCTCGTCCAGGCGGTCGCGGGTCCG	3469
	CCAAGCTG**G**AGGAGCAG	3470
	CTGCTCCT**C**CAGCTTGG	3471

EXAMPLE 21
Familial hypercholesterolemia - LDLR

Familial hypercholesterolemia is characterized by elevation of serum cholesterol bound to low density lipoprotein (LDL) and is, hence, one of the conditions producing a hyperlipoproteinemia phenotype. Familial hypercholesterolemia is an autosomal dominant disorder characterized by elevation

of serum cholesterol bound to low density lipoprotein (LDL). Mutations in the LDL receptor (LDLR) gene cause this disorder. The attached table discloses the correcting oligonucleotide base sequences for the LDLR oligonucleotides of the invention.

Table 28
LDLR Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Hypercholesterolaemia Glu10Term cGAG-TAG	GCGTTGAGAGACCCTTTCTCCTTTTCCTCTCTCTCAGTGGGC GACAGATGCGAAAGAAACGAGTTCCAGTGCCAAGACGGGAA ATGCATCTCCTACAAGTGGGTCTGCGATGGCAGCGCTG	3472
	CAGCGCTGCCATCGCAGACCCACTTGTAGGAGATGCATTTCC CGTCTTGGCACTGGAACTCGTTTCTTTCGCATCTGTCGCCCA CTGAGAGAGAGGAAAAGGAGAAAGGGTCTCTCAACGC	3473
	AAAGAAACGAGTTCCAG	3474
	CTGGAACTCGTTTCTTT	3475
Hypercholesterolaemia Gln12Term cCAG-TAG	AGAGACCCTTTCTCCTTTTCCTCTCTCTCAGTGGGCGACAGA TGCGAAAGAAACGAGTTCCAGTGCCAAGACGGGAAATGCATC TCCTACAAGTGGGTCTGCGATGGCAGCGCTGAGTGCC	3476
	GGCACTCAGCGCTGCCATCGCAGACCCACTTGTAGGAGATG CATTTCCCGTCTTGGCACTGGAACTCGTTTCTTTCGCATCTGT CGCCCACTGAGAGAGAGGAAAAGGAGAAAGGGTCTCT	3477
	ACGAGTTCCAGTGCCAA	3478
	TTGGCACTGGAACTCGT	3479
Hypercholesterolaemia Gln14Term cCAA-TAA	CCTTTCTCCTTTTCCTCTCTCTCAGTGGGCGACAGATGCGAA AGAAACGAGTTCCAGTGCCAAGACGGGAAATGCATCTCCTAC AAGTGGGTCTGCGATGGCAGCGCTGAGTGCCAGGATG	3480
	CATCCTGGCACTCAGCGCTGCCATCGCAGACCCACTTGTAG GAGATGCATTTCCCGTCTTGGCACTGGAACTCGTTTCTTTCG CATCTGTCGCCCACTGAGAGAGAGGAAAAGGAGAAAGG	3481
	TCCAGTGCCAAGACGGG	3482
	CCCGTCTTGGCACTGGA	3483
Hypercholesterolaemia Trp23Term TGG-TAG	GCGACAGATGCGAAAGAAACGAGTTCCAGTGCCAAGACGGG AAATGCATCTCCTACAAGTGGGTCTGCGATGGCAGCGCTGAG TGCCAGGATGGCTCTGATGAGTCCCAGGAGACGTGCTG	3484
	CAGCACGTCTCCTGGGACTCATCAGAGCCATCCTGGCACTCA GCGCTGCCATCGCAGACCCACTTGTAGGAGATGCATTTCCCG TCTTGGCACTGGAACTCGTTTCTTTCGCATCTGTCGC	3485
	CTACAAGTGGGTCTGCG	3486
	CGCAGACCCACTTGTAG	3487
Hypercholesterolaemia Ala29Ser cGCT-TCT	AACGAGTTCCAGTGCCAAGACGGGAAATGCATCTCCTACAAG TGGGTCTGCGATGGCAGCGCTGAGTGCCAGGATGGCTCTGA TGAGTCCCAGGAGACGTGCTGTGAGTCCCCTTTGGGCA	3488

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGCCCAAAGGGGACTCACAGCACGTCTCCTGGGACTCATCA GAGCCATCCTGGCACTCAGCGCTGCCATCGCAGACCCACTT GTAGGAGATGCATTTCCCGTCTTGGCACTGGAACTCGTT	3489
	ATGGCAGCGCTGAGTGC	3490
	GCACTCAGCGCTGCCAT	3491
Hypercholesterolaemia Cys31Tyr TGC-TAC	TCCAGTGCCAAGACGGGAAATGCATCTCCTACAAGTGGGTCT GCGATGGCAGCGCTGAGTGCCAGGATGGCTCTGATGAGTCC CAGGAGACGTGCTGTGAGTCCCCTTTGGGCATGATATG	3492
	CATATCATGCCCAAAGGGGACTCACAGCACGTCTCCTGGGAC TCATCAGAGCCATCCTGGCACTCAGCGCTGCCATCGCAGAC CCACTTGTAGGAGATGCATTTCCCGTCTTGGCACTGGA	3493
	CGCTGAGTGCCAGGATG	3494
	CATCCTGGCACTCAGCG	3495
Hypercholesterolaemia Arg57Cys cCGT-TGT	AATCCTGTCTCTTCTGTAGTGTCTGTCACCTGCAAATCCGGG GACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCATTCCTCA GTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACAACG	3496
	CGTTGTCGCAGTCCACTTGGCCATCGCACCTCCAGAACTGAG GAATGCAGCGGTTGACACGGCCCCCACAGCTGAAGTCCCCG GATTTGCAGGTGACAGACACTACAGAAGAGACAGGATT	3497
	GTGGGGGCCGTGTCAAC	3498
	GTTGACACGGCCCCCAC	3499
Hypercholesterolaemia Gln64Term tCAG-TAG	TCTGTCACCTGCAAATCCGGGGACTTCAGCTGTGGGGGCCG TGTCAACCGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCA AGTGGACTGCGACAACGGCTCAGACGAGCAAGGCTGTC	3500
	GACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCCACTTGGC CATCGCACCTCCAGAACTGAGGAATGCAGCGGTTGACACGG CCCCCACAGCTGAAGTCCCCGGATTTGCAGGTGACAGA	3501
	GCATTCCTCAGTTCTGG	3502
	CCAGAACTGAGGAATGC	3503
Hypercholesterolaemia Trp66Gly cTGG-GGG	ACCTGCAAATCCGGGGACTTCAGCTGTGGGGGCCGTGTCAA CCGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGG ACTGCGACAACGGCTCAGACGAGCAAGGCTGTCGTAAGT	3504
	ACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCCA CTTGGCCATCGCACCTCCAGAACTGAGGAATGCAGCGGTTG ACACGGCCCCCACAGCTGAAGTCCCCGGATTTGCAGGT	3505
	CTCAGTTCTGGAGGTGC	3506
	GCACCTCCAGAACTGAG	3507
Hypercholesterolaemia Trp66Term TGG-TAG	CCTGCAAATCCGGGGACTTCAGCTGTGGGGGCCGTGTCAAC CGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGA CTGCGACAACGGCTCAGACGAGCAAGGCTGTCGTAAGTG	3508
	CACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCC ACTTGGCCATCGCACCTCCAGAACTGAGGAATGCAGCGGTTG ACACGGCCCCCACAGCTGAAGTCCCCGGATTTGCAGG	3509
	TCAGTTCTGGAGGTGCG	3510

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CGCACCTCCAGAACTGA	3511
Hypercholesterolaemia Cys68Arg gTGC-CGC	AAATCCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTG CATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGA CAACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCC	3512
	GGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGC AGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAATGCAG CGGTTGACACGGCCCCCACAGCTGAAGTCCCCGGATTT	3513
	TCTGGAGGTGCGATGGC	3514
	GCCATCGCACCTCCAGA	3515
Hypercholesterolaemia Cys68Trp TGCg-TGG	ATCCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCA TTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACA ACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCCT	3516
	AGGGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTC GCAGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAATGCA GCGGTTGACACGGCCCCCACAGCTGAAGTCCCCGGAT	3517
	TGGAGGTGCGATGGCCA	3518
	TGGCCATCGCACCTCCA	3519
Hypercholesterolaemia Cys68Tyr TGC-TAC	AATCCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTGC ATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGAC AACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCC	3520
	GGGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTC GCAGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAATGCA GCGGTTGACACGGCCCCCACAGCTGAAGTCCCCGGATT	3521
	CTGGAGGTGCGATGGCC	3522
	GGCCATCGCACCTCCAG	3523
Hypercholesterolaemia Asp69Asn cGAT-AAT	TCCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCAT TCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACA ACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCCTG	3524
	CAGGGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGT CGCAGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAATG CAGCGGTTGACACGGCCCCCACAGCTGAAGTCCCCGGA	3525
	GGAGGTGCGATGGCCAA	3526
	TTGGCCATCGCACCTCC	3527
Hypercholesterolaemia Asp69Gly GAT-GGT	CCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCATT CCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACAA CGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCCTGC	3528
	GCAGGGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTT GTCGCAGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAAT GCAGCGGTTGACACGGCCCCCACAGCTGAAGTCCCCGG	3529
	GAGGTGCGATGGCCAAG	3530
	CTTGGCCATCGCACCTC	3531
Hypercholesterolaemia Asp69Tyr cGAT-TAT	TCCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCAT TCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACA ACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCCTG	3532

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CAGGGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGT CGCAGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAATG CAGCGGTTGACACGGCCCCCACAGCTGAAGTCCCCGGA	3533
	GGAGGTGCGATGGCCAA	3534
	TTGGCCATCGCACCTCC	3535
Hypercholesterolaemia Gln71Glu cCAA-GAA	GACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCATTCCTCA GTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACAACGGCT CAGACGAGCAAGGCTGTCGTAAGTGTGGCCCTGCCTTTG	3536
	CAAAGGCAGGGCCACACTTACGACAGCCTTGCTCGTCTGAG CCGTTGTCGCAGTCCACTTGGCCATCGCACCTCCAGAACTGA GGAATGCAGCGGTTGACACGGCCCCCACAGCTGAAGTC	3537
	GCGATGGCCAAGTGGAC	3538
	GTCCACTTGGCCATCGC	3539
Hypercholesterolaemia Cys74Gly cTGC-GGC	TGTGGGGGCCGTGTCAACCGCTGCATTCCTCAGTTCTGGAG GTGCGATGGCCAAGTGGACTGCGACAACGGCTCAGACGAGC AAGGCTGTCGTAAGTGTGGCCCTGCCTTTGCTATTGAGC	3540
	GCTCAATAGCAAAGGCAGGGCCACACTTACGACAGCCTTGCT CGTCTGAGCCGTTGTCGCAGTCCACTTGGCCATCGCACCTC CAGAACTGAGGAATGCAGCGGTTGACACGGCCCCCACA	3541
	AAGTGGACTGCGACAAC	3542
	GTTGTCGCAGTCCACTT	3543
Hypercholesterolaemia Ser78Term TCA-TGA	TCAACCGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAG TGGACTGCGACAACGGCTCAGACGAGCAAGGCTGTCGTAAG TGTGGCCCTGCCTTTGCTATTGAGCCTATCTGAGTCCT	3544
	AGGACTCAGATAGGCTCAATAGCAAAGGCAGGGCCACACTTA CGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCCACTTGG CCATCGCACCTCCAGAACTGAGGAATGCAGCGGTTGA	3545
	CAACGGCTCAGACGAGC	3546
	GCTCGTCTGAGCCGTTG	3547
Hypercholesterolaemia Glu80Lys cGAG-AAG	CGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGA CTGCGACAACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTG GCCCTGCCTTTGCTATTGAGCCTATCTGAGTCCTGGGGA	3548
	TCCCCAGGACTCAGATAGGCTCAATAGCAAAGGCAGGGCCA CACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCC ACTTGGCCATCGCACCTCCAGAACTGAGGAATGCAGCG	3549
	GCTCAGACGAGCAAGGC	3550
	GCCTTGCTCGTCTGAGC	3551
Hypercholesterolaemia Glu80Term cGAG-TAG	CGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGA CTGCGACAACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTG GCCCTGCCTTTGCTATTGAGCCTATCTGAGTCCTGGGGA	3552
	TCCCCAGGACTCAGATAGGCTCAATAGCAAAGGCAGGGCCA CACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCC ACTTGGCCATCGCACCTCCAGAACTGAGGAATGCAGCG	3553
	GCTCAGACGAGCAAGGC	3554

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCCTTGCTCGTCTGAGC	3555
Hypercholesterolaemia Gln81Term gCAA-TAA	TGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGC GACAACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCC TGCCTTTGCTATTGAGCCTATCTGAGTCCTGGGGAGTG	3556
	CACTCCCCAGGACTCAGATAGGCTCAATAGCAAAGGCAGGG CCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAG TCCACTTGGCCATCGCACCTCCAGAACTGAGGAATGCA	3557
	CAGACGAGCAAGGCTGT	3558
	ACAGCCTTGCTCGTCTG	3559
Hypercholesterolaemia Cys88Arg gTGC-CGC	TGGGAGACTTCACACGGTGATGGTGGTCTCGGCCCATCCAT CCCTGCAGCCCCCAAGACGTGCTCCCAGGACGAGTTTCGCT GCCACGATGGGAAGTGCATCTCTCGGCAGTTCGTCTGTG	3560
	CACAGACGAACTGCCGAGAGATGCACTTCCCATCGTGGCAG CGAAACTCGTCCTGGGAGCACGTCTTGGGGGCTGCAGGGAT GGATGGGCCGAGACCACCATCACCGTGTGAAGTCTCCCA	3561
	CCAAGACGTGCTCCCAG	3562
	CTGGGAGCACGTCTTGG	3563
Hypercholesterolaemia Glu92Term cGAG-TAG	CACGGTGATGGTGGTCTCGGCCCATCCATCCCTGCAGCCCC CAAGACGTGCTCCCAGGACGAGTTTCGCTGCCACGATGGGA AGTGCATCTCTCGGCAGTTCGTCTGTGACTCAGACCGGG	3564
	CCCGGTCTGAGTCACAGACGAACTGCCGAGAGATGCACTTC CCATCGTGGCAGCGAAACTCGTCCTGGGAGCACGTCTTGGG GGCTGCAGGGATGGATGGGCCGAGACCACCATCACCGTG	3565
	CCCAGGACGAGTTTCGC	3566
	GCGAAACTCGTCCTGGG	3567
Hypercholesterolaemia Cys95Arg cTGC-CGC	GGTGGTCTCGGCCCATCCATCCCTGCAGCCCCCAAGACGTG CTCCCAGGACGAGTTTCGCTGCCACGATGGGAAGTGCATCT CTCGGCAGTTCGTCTGTGACTCAGACCGGGACTGCTTGG	3568
	CCAAGCAGTCCCGGTCTGAGTCACAGACGAACTGCCGAGAG ATGCACTTCCCATCGTGGCAGCGAAACTCGTCCTGGGAGCA CGTCTTGGGGGCTGCAGGGATGGATGGGCCGAGACCACC	3569
	AGTTTCGCTGCCACGAT	3570
	ATCGTGGCAGCGAAACT	3571
Hypercholesterolaemia Asp97Tyr cGAT-TAT	CTCGGCCCATCCATCCCTGCAGCCCCCAAGACGTGCTCCCA GGACGAGTTTCGCTGCCACGATGGGAAGTGCATCTCTCGGC AGTTCGTCTGTGACTCAGACCGGGACTGCTTGGACGGCT	3572
	AGCCGTCCAAGCAGTCCCGGTCTGAGTCACAGACGAACTGC CGAGAGATGCACTTCCCATCGTGGCAGCGAAACTCGTCCTG GGAGCACGTCTTGGGGGCTGCAGGGATGGATGGGCCGAG	3573
	GCTGCCACGATGGGAAG	3574
	CTTCCCATCGTGGCAGC	3575
Hypercholesterolaemia Trp(-12)Arg cTGG-AGG	GGGTCGGGACACTGCCTGGCAGAGGCTGCGAGCATGGGGC CCTGGGGCTGGAAATTGCGCTGGACCGTCGCCTTGCTCCTC GCCGCGGCGGGGACTGCAGGTAAGGCTTGCTCCAGGCGCC	3576

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GGCGCCTGGAGCAAGCCTTACCTGCAGTCCCCGCCGCGGC GAGGAGCAAGGCGACGGTCCAGCGCAATTTCCAGCCCCAGG GCCCCATGCTCGCAGCCTCTGCCAGGCAGTGTCCCGACCC	3577
	AATTGCGCTGGACCGTC	3578
	GACGGTCCAGCGCAATT	3579
Hypercholesterolaemia Trp(-18)Term TGGg-TGA	CAGCAGGTCGTGATCCGGGTCGGGACACTGCCTGGCAGAGG CTGCGAGCATGGGGCCCTGGGGCTGGAAATTGCGCTGGACC GTCGCCTTGCTCCTCGCCGCGGCGGGGACTGCAGGTAAG	3580
	CTTACCTGCAGTCCCCGCCGCGGCGAGGAGCAAGGCGACG GTCCAGCGCAATTTCCAGCCCCAGGGCCCCATGCTCGCAGC CTCTGCCAGGCAGTGTCCCGACCCGGATCACGACCTGCTG	3581
	GGGCCCTGGGGCTGGAA	3582
	TTCCAGCCCCAGGGCCC	3583
Hypercholesterolaemia Met(-21)Leu cATG-TTG	CAGCTAGGACACAGCAGGTCGTGATCCGGGTCGGGACACTG CCTGGCAGAGGCTGCGAGCATGGGGCCCTGGGGCTGGAAA TTGCGCTGGACCGTCGCCTTGCTCCTCGCCGCGGCGGGGA	3584
	TCCCCGCCGCGGCGAGGAGCAAGGCGACGGTCCAGCGCAA TTTCCAGCCCCAGGGCCCCATGCTCGCAGCCTCTGCCAGGC AGTGTCCCGACCCGGATCACGACCTGCTGTGTCCTAGCTG	3585
	CTGCGAGCATGGGGCCC	3586
	GGGCCCCATGCTCGCAG	3587
Hypercholesterolaemia Met(-21)Val cATG-GTG	CAGCTAGGACACAGCAGGTCGTGATCCGGGTCGGGACACTG CCTGGCAGAGGCTGCGAGCATGGGGCCCTGGGGCTGGAAA TTGCGCTGGACCGTCGCCTTGCTCCTCGCCGCGGCGGGGA	3588
	TCCCCGCCGCGGCGAGGAGCAAGGCGACGGTCCAGCGCAA TTTCCAGCCCCAGGGCCCCATGCTCGCAGCCTCTGCCAGGC AGTGTCCCGACCCGGATCACGACCTGCTGTGTCCTAGCTG	3589
	CTGCGAGCATGGGGCCC	3590
	GGGCCCCATGCTCGCAG	3591
Hypercholesterolaemia Ile101Phe cATC-TTC	ATCCCTGCAGCCCCCAAGACGTGCTCCCAGGACGAGTTTCG CTGCCACGATGGGAAGTGCATCTCTCGGCAGTTCGTCTGTGA CTCAGACCGGGACTGCTTGGACGGCTCAGACGAGGCCT	3592
	AGGCCTCGTCTGAGCCGTCCAAGCAGTCCCGGTCTGAGTCA CAGACGAACTGCCGAGAGATGCACTTCCCATCGTGGCAGCG AAACTCGTCCTGGGAGCACGTCTTGGGGGCTGCAGGGAT	3593
	GGAAGTGCATCTCTCGG	3594
	CCGAGAGATGCACTTCC	3595
Hypercholesterolaemia Gln104Term gCAG-TAG	GCCCCCAAGACGTGCTCCCAGGACGAGTTTCGCTGCCACGA TGGGAAGTGCATCTCTCGGCAGTTCGTCTGTGACTCAGACCG GGACTGCTTGGACGGCTCAGACGAGGCCTCCTGCCCGG	3596
	CCGGGCAGGAGGCCTCGTCTGAGCCGTCCAAGCAGTCCCG GTCTGAGTCACAGACGAACTGCCGAGAGATGCACTTCCCATC GTGGCAGCGAAACTCGTCCTGGGAGCACGTCTTGGGGGC	3597
	TCTCTCGGCAGTTCGTC	3598

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GACGAACTGCCGAGAGA	3599
Hypercholesterolaemia Cys113Arg cTGC-CGC	TTTCGCTGCCACGATGGGAAGTGCATCTCTCGGCAGTTCGTC TGTGACTCAGACCGGGACTGCTTGGACGGCTCAGACGAGGC CTCCTGCCCGGTGCTCACCTGTGGTCCCGCCAGCTTCC	3600
	GGAAGCTGGCGGGACCACAGGTGAGCACCGGGCAGGAGGC CTCGTCTGAGCCGTCCAAGCAGTCCCGGTCTGAGTCACAGA CGAACTGCCGAGAGATGCACTTCCCATCGTGGCAGCGAAA	3601
	ACCGGGACTGCTTGGAC	3602
	GTCCAAGCAGTCCCGGT	3603
Hypercholesterolaemia Glu119Lys cGAG-AAG	AAGTGCATCTCTCGGCAGTTCGTCTGTGACTCAGACCGGGAC TGCTTGGACGGCTCAGACGAGGCCTCCTGCCCGGTGCTCAC CTGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCT	3604
	AGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACAGGTG AGCACCGGGCAGGAGGCCTCGTCTGAGCCGTCCAAGCAGTC CCGGTCTGAGTCACAGACGAACTGCCGAGAGATGCACTT	3605
	GCTCAGACGAGGCCTCC	3606
	GGAGGCCTCGTCTGAGC	3607
Hypercholesterolaemia Glu119Term cGAG-TAG	AAGTGCATCTCTCGGCAGTTCGTCTGTGACTCAGACCGGGAC TGCTTGGACGGCTCAGACGAGGCCTCCTGCCCGGTGCTCAC CTGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCT	3608
	AGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACAGGTG AGCACCGGGCAGGAGGCCTCGTCTGAGCCGTCCAAGCAGTC CCGGTCTGAGTCACAGACGAACTGCCGAGAGATGCACTT	3609
	GCTCAGACGAGGCCTCC	3610
	GGAGGCCTCGTCTGAGC	3611
Hypercholesterolaemia Cys122Term TGCc-TGA	TCGGCAGTTCGTCTGTGACTCAGACCGGGACTGCTTGGACG GCTCAGACGAGGCCTCCTGCCCGGTGCTCACCTGTGGTCCC GCCAGCTTCCAGTGCAACAGCTCCACCTGCATCCCCCAG	3612
	CTGGGGGATGCAGGTGGAGCTGTTGCACTGGAAGCTGGCGG GACCACAGGTGAGCACCGGGCAGGAGGCCTCGTCTGAGCC GTCCAAGCAGTCCCGGTCTGAGTCACAGACGAACTGCCGA	3613
	GCCTCCTGCCCGGTGCT	3614
	AGCACCGGGCAGGAGGC	3615
Hypercholesterolaemia Cys127Trp TGTg-TGG	TGACTCAGACCGGGACTGCTTGGACGGCTCAGACGAGGCCT CCTGCCCGGTGCTCACCTGTGGTCCCGCCAGCTTCCAGTGC AACAGCTCCACCTGCATCCCCCAGCTGTGGGCCTGCGAC	3616
	GTCGCAGGCCCACAGCTGGGGGATGCAGGTGGAGCTGTTGC ACTGGAAGCTGGCGGGACCACAGGTGAGCACCGGGCAGGA GGCCTCGTCTGAGCCGTCCAAGCAGTCCCGGTCTGAGTCA	3617
	CTCACCTGTGGTCCCGC	3618
	GCGGGACCACAGGTGAG	3619
Hypercholesterolaemia Gln133Term cCAG-TAG	TGCTTGGACGGCTCAGACGAGGCCTCCTGCCCGGTGCTCAC CTGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCAT CCCCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCG	3620

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CGCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGGAT GCAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACAGG TGAGCACCGGGCAGGAGGCCTCGTCTGAGCCGTCCAAGCA	3621
	CCAGCTTCCAGTGCAAC	3622
	GTTGCACTGGAAGCTGG	3623
Hypercholesterolaemia Cys134Gly gTGC-GGC	TTGGACGGCTCAGACGAGGCCTCCTGCCCGGTGCTCACCTG TGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCATCC CCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCGAAG	3624
	CTTCGCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGG ATGCAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACA GGTGAGCACCGGGCAGGAGGCCTCGTCTGAGCCGTCCAA	3625
	GCTTCCAGTGCAACAGC	3626
	GCTGTTGCACTGGAAGC	3627
Hypercholesterolaemia Cys139Gly cTGC-GGC	GAGGCCTCCTGCCCGGTGCTCACCTGTGGTCCCGCCAGCTT CCAGTGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGCCT GCGACAACGACCCCGACTGCGAAGATGGCTCGGATGAGT	3628
	ACTCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCGCAG GCCCACAGCTGGGGGATGCAGGTGGAGCTGTTGCACTGGAA GCTGGCGGGACCACAGGTGAGCACCGGGCAGGAGGCCTC	3629
	GCTCCACCTGCATCCCC	3630
	GGGGATGCAGGTGGAGC	3631
Hypercholesterolaemia Cys139Tyr TGC-TAC	AGGCCTCCTGCCCGGTGCTCACCTGTGGTCCCGCCAGCTTC CAGTGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGCCTG CGACAACGACCCCGACTGCGAAGATGGCTCGGATGAGTG	3632
	CACTCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCGCA GGCCCACAGCTGGGGGATGCAGGTGGAGCTGTTGCACTGGA AGCTGGCGGGACCACAGGTGAGCACCGGGCAGGAGGCCT	3633
	CTCCACCTGCATCCCCC	3634
	GGGGGATGCAGGTGGAG	3635
Hypercholesterolaemia Cys146Term TGCg-TGA	CTGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCAT CCCCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCGAAG ATGGCTCGGATGAGTGGCCGCAGCGCTGTAGGGGTCTT	3636
	AAGACCCCTACAGCGCTGCGGCCACTCATCCGAGCCATCTTC GCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGGATG CAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACAG	3637
	TGGGCCTGCGACAACGA	3638
	TCGTTGTCGCAGGCCCA	3639
Hypercholesterolaemia Asp147Asn cGAC-AAC	TGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCATC CCCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCGAAGA TGGCTCGGATGAGTGGCCGCAGCGCTGTAGGGGTCTTT	3640
	AAAGACCCCTACAGCGCTGCGGCCACTCATCCGAGCCATCTT CGCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGGAT GCAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACA	3641
	GGGCCTGCGACAACGAC	3642

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GTCGTTGTCGCAGGCCC	3643
Hypercholesterolaemia Asp147His cGAC-CAC	TGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCATC CCCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCGAAGA TGGCTCGGATGAGTGGCCGCAGCGCTGTAGGGGTCTTT	3644
	AAAGACCCCTACAGCGCTGCGGCCACTCATCCGAGCCATCTT CGCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGGAT GCAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACA	3645
	GGGCCTGCGACAACGAC	3646
	GTCGTTGTCGCAGGCCC	3647
Hypercholesterolaemia Asp147Tyr cGAC-TAC	TGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCATC CCCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCGAAGA TGGCTCGGATGAGTGGCCGCAGCGCTGTAGGGGTCTTT	3648
	AAAGACCCCTACAGCGCTGCGGCCACTCATCCGAGCCATCTT CGCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGGAT GCAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACA	3649
	GGGCCTGCGACAACGAC	3650
	GTCGTTGTCGCAGGCCC	3651
Hypercholesterolaemia Cys152Arg cTGC-CGC	TTCCAGTGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGC CTGCGACAACGACCCCGACTGCGAAGATGGCTCGGATGAGT GGCCGCAGCGCTGTAGGGGTCTTTACGTGTTCCAAGGGG	3652
	CCCCTTGGAACACGTAAAGACCCCTACAGCGCTGCGGCCAC TCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCGCAGGC CCACAGCTGGGGGATGCAGGTGGAGCTGTTGCACTGGAA	3653
	ACCCCGACTGCGAAGAT	3654
	ATCTTCGCAGTCGGGGT	3655
Hypercholesterolaemia Cys152Gly cTGC-GGC	TTCCAGTGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGC CTGCGACAACGACCCCGACTGCGAAGATGGCTCGGATGAGT GGCCGCAGCGCTGTAGGGGTCTTTACGTGTTCCAAGGGG	3656
	CCCCTTGGAACACGTAAAGACCCCTACAGCGCTGCGGCCAC TCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCGCAGGC CCACAGCTGGGGGATGCAGGTGGAGCTGTTGCACTGGAA	3657
	ACCCCGACTGCGAAGAT	3658
	ATCTTCGCAGTCGGGGT	3659
Hypercholesterolaemia Cys152Trp TGCg-TGG	CCAGTGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGCCT GCGACAACGACCCCGACTGCGAAGATGGCTCGGATGAGTGG CCGCAGCGCTGTAGGGGTCTTTACGTGTTCCAAGGGGAC	3660
	GTCCCCTTGGAACACGTAAAGACCCCTACAGCGCTGCGGCC ACTCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCGCAG GCCCACAGCTGGGGGATGCAGGTGGAGCTGTTGCACTGG	3661
	CCCGACTGCGAAGATGG	3662
	CCATCTTCGCAGTCGGG	3663
Hypercholesterolaemia Asp154Asn aGAT-AAT	TGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGCCTGCGA CAACGACCCCGACTGCGAAGATGGCTCGGATGAGTGGCCGC AGCGCTGTAGGGGTCTTTACGTGTTCCAAGGGGACAGTA	3664

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TACTGTCCCCTTGGAACACGTAAAGACCCCTACAGCGCTGCG GCCACTCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCG CAGGCCCACAGCTGGGGGATGCAGGTGGAGCTGTTGCA	3665
	ACTGCGAAGATGGCTCG	3666
	CGAGCCATCTTCGCAGT	3667
Hypercholesterolaemia Ser156Leu TCG-TTG	GCTCCACCTGCATCCCCCAGCTGTGGGCCTGCGACAACGAC CCCGACTGCGAAGATGGCTCGGATGAGTGGCCGCAGCGCTG TAGGGGTCTTTACGTGTTCCAAGGGGACAGTAGCCCCTG	3668
	CAGGGGCTACTGTCCCCTTGGAACACGTAAAGACCCCTACAG CGCTGCGGCCACTCATCCGAGCCATCTTCGCAGTCGGGGTC GTTGTCGCAGGCCCACAGCTGGGGGATGCAGGTGGAGC	3669
	AGATGGCTCGGATGAGT	3670
	ACTCATCCGAGCCATCT	3671
Hypercholesterolaemia Cys163Tyr TGT-TAT	TGTGGGCCTGCGACAACGACCCCGACTGCGAAGATGGCTCG GATGAGTGGCCGCAGCGCTGTAGGGGTCTTTACGTGTTCCAA GGGGACAGTAGCCCCTGCTCGGCCTTCGAGTTCCACTG	3672
	CAGTGGAACTCGAAGGCCGAGCAGGGGCTACTGTCCCCTTG GAACACGTAAAGACCCCTACAGCGCTGCGGCCACTCATCCG AGCCATCTTCGCAGTCGGGGTCGTTGTCGCAGGCCCACA	3673
	GCAGCGCTGTAGGGGTC	3674
	GACCCCTACAGCGCTGC	3675
Hypercholesterolaemia Tyr167Term TACg-TAG	CAACGACCCCGACTGCGAAGATGGCTCGGATGAGTGGCCGC AGCGCTGTAGGGGTCTTTACGTGTTCCAAGGGGACAGTAGC CCCTGCTCGGCCTTCGAGTTCCACTGCCTAAGTGGCGAG	3676
	CTCGCCACTTAGGCAGTGGAACTCGAAGGCCGAGCAGGGGC TACTGTCCCCTTGGAACACGTAAAGACCCCTACAGCGCTGCG GCCACTCATCCGAGCCATCTTCGCAGTCGGGGTCGTTG	3677
	GGTCTTTACGTGTTCCA	3678
	TGGAACACGTAAAGACC	3679
Hypercholesterolaemia Gln170Term cCAA-TAA	CCCGACTGCGAAGATGGCTCGGATGAGTGGCCGCAGCGCTG TAGGGGTCTTTACGTGTTCCAAGGGGACAGTAGCCCCTGCTC GGCCTTCGAGTTCCACTGCCTAAGTGGCGAGTGCATCC	3680
	GGATGCACTCGCCACTTAGGCAGTGGAACTCGAAGGCCGAG CAGGGGCTACTGTCCCCTTGGAACACGTAAAGACCCCTACAG CGCTGCGGCCACTCATCCGAGCCATCTTCGCAGTCGGG	3681
	ACGTGTTCCAAGGGGAC	3682
	GTCCCCTTGGAACACGT	3683
Hypercholesterolaemia Cys176Phe TGC-TTC	CGGATGAGTGGCCGCAGCGCTGTAGGGGTCTTTACGTGTTC CAAGGGGACAGTAGCCCCTGCTCGGCCTTCGAGTTCCACTG CCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGA	3684
	TCACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGCA GTGGAACTCGAAGGCCCAGCAGGGGCTACTGTCCCCTTGGA ACACGTAAAGACCCCTACAGCGCTGCGGCCACTCATCCG	3685
	TAGCCCCTGCTCGGCCT	3686

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGGCCGAGCAGGGGCTA	3687
Hypercholesterolaemia Cys176Tyr TGC-TAC	CGGATGAGTGGCCGCAGCGCTGTAGGGGTCTTTACGTGTTC CAAGGGGACAGTAGCCCCTGCTCGGCCTTCGAGTTCCACTG CCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGA	3688
	TCACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGCA GTGGAACTCGAAGGCCGAGCAGGGGCTACTGTCCCCTTGGA ACACGTAAAGACCCCTACAGCGCTGCGGCCACTCATCCG	3689
	TAGCCCCTGCTCGGCCT	3690
	AGGCCGAGCAGGGGCTA	3691
Hypercholesterolaemia Ser177Leu TCG-TTG	ATGAGTGGCCGCAGCGCTGTAGGGGTCTTTACGTGTTCCAAG GGGACAGTAGCCCCTGCTCGGCCTTCGAGTTCCACTGCCTA AGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGATGG	3692
	CCATCACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAG GCAGTGGAACTCGAAGGCCGAGCAGGGGCTACTGTCCCCTT GGAACACGTAAAGACCCCTACAGCGCTGCGGCCACTCAT	3693
	CCCCTGCTCGGCCTTCG	3694
	CGAAGGCCGAGCAGGGG	3695
Hypercholesterolaemia Glu187Lys cGAG-AAG	TACGTGTTCCAAGGGGACAGTAGCCCCTGCTCGGCCTTCGA GTTCCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTGGC GCTGTGATGGTGGCCCCGACTGCAAGGACAAATCTGACG	3696
	CGTCAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGCGC CAGCTGGAGTGGATGCACTCGCCACTTAGGCAGTGGAACTC GAAGGCCGAGCAGGGGCTACTGTCCCCTTGGAACACGTA	3697
	TAAGTGGCGAGTGCATC	3698
	GATGCACTCGCCACTTA	3699
Hypercholesterolaemia His190Tyr cCAC-TAC	CAAGGGGACAGTAGCCCCTGCTCGGCCTTCGAGTTCCACTG CCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGATG GTGGCCCCGACTGCAAGGACAAATCTGACGAGGAAAACT	3700
	AGTTTTCCTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCAT CACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGCAG TGGAACTCGAAGGCCGAGCAGGGGCTACTGTCCCCTTG	3701
	AGTGCATCCACTCCAGC	3702
	GCTGGAGTGGATGCACT	3703
Hypercholesterolaemia Gly198Asp GGC-GAC	CCTTCGAGTTCCACTGCCTAAGTGGCGAGTGCATCCACTCCA GCTGGCGCTGTGATGGTGGCCCCGACTGCAAGGACAAATCT GACGAGGAAAACTGCGGTATGGGCGGGGCCAGGGTGGG	3704
	CCCACCCTGGCCCCGCCCATACCGCAGTTTTCCTCGTCAGAT TTGTCCTTGCAGTCGGGGCCACCATCACAGCGCCAGCTGGA GTGGATGCACTCGCCACTTAGGCAGTGGAACTCGAAGG	3705
	TGATGGTGGCCCCGACT	3706
	AGTCGGGGCCACCATCA	3707
Hypercholesterolaemia Asp200Asn cGAC-AAC	GAGTTCCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTG GCGCTGTGATGGTGGCCCCGACTGCAAGGACAAATCTGACG AGGAAAACTGCGGTATGGGCGGGGCCAGGGTGGGGGCGG	3708

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCGCCCCCACCCTGGCCCCGCCCATACCGCAGTTTTCCTCG TCAGATTTGTCCTTGCAGTCGGGGGCCACCATCACAGCGCCAG CTGGAGTGGATGCACTCGCCACTTAGGCAGTGGAACTC	3709
	GTGGCCCCGACTGCAAG	3710
	CTTGCAGTCGGGGCCAC	3711
Hypercholesterolaemia Asp200Gly GAC-GGC	AGTTCCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTGGC GCTGTGATGGTGGCCCCGACTGCAAGGACAAATCTGACGAG GAAAACTGCGGTATGGGCGGGGCCAGGGTGGGGGCGGG	3712
	CCCGCCCCCACCCTGGCCCCGCCCATACCGCAGTTTTCCTC GTCAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGCGCCA GCTGGAGTGGATGCACTCGCCACTTAGGCAGTGGAACT	3713
	TGGCCCCGACTGCAAGG	3714
	CCTTGCAGTCGGGGCCA	3715
Hypercholesterolaemia Asp200Tyr cGAC-TAC	GAGTTCCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTG GCGCTGTGATGGTGGCCCCGACTGCAAGGACAAATCTGACG AGGAAAACTGCGGTATGGGCGGGGCCAGGGTGGGGGCGG	3716
	CCGCCCCCACCCTGGCCCCGCCCATACCGCAGTTTTCCTCG TCAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGCGCCAG CTGGAGTGGATGCACTCGCCACTTAGGCAGTGGAACTC	3717
	GTGGCCCCGACTGCAAG	3718
	CTTGCAGTCGGGGCCAC	3719
Hypercholesterolaemia Cys201Term TGCa-TGA	CCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCT GTGATGGTGGCCCCGACTGCAAGGACAAATCTGACGAGGAA AACTGCGGTATGGGCGGGGCCAGGGTGGGGGCGGGGCGT	3720
	ACGCCCCGCCCCCACCCTGGCCCCGCCCATACCGCAGTTTT CCTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGC GCCAGCTGGAGTGGATGCACTCGCCACTTAGGCAGTGG	3721
	CCCGACTGCAAGGACAA	3722
	TTGTCCTTGCAGTCGGG	3723
Hypercholesterolaemia Cys201Tyr TGC-TAC	TCCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGC TGTGATGGTGGCCCCGACTGCAAGGACAAATCTGACGAGGA AAACTGCGGTATGGGCGGGGCCAGGGTGGGGGCGGGGCG	3724
	CGCCCCGCCCCCACCCTGGCCCCGCCCATACCGCAGTTTTC CTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGCG CCAGCTGGAGTGGATGCACTCGCCACTTAGGCAGTGGA	3725
	CCCCGACTGCAAGGACA	3726
	TGTCCTTGCAGTCGGGG	3727
Hypercholesterolaemia Asp203Asn gGAC-AAC	TGCCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGA TGGTGGCCCCGACTGCAAGGACAAATCTGACGAGGAAAACT GCGGTATGGGCGGGGCCAGGGTGGGGGCGGGGCGTCCTA	3728
	TAGGACGCCCCGCCCCCACCCTGGCCCCGCCCATACCGCA GTTTTCCTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCATC ACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGCA	3729
	ACTGCAAGGACAAATCT	3730

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGATTTGTCCTTGCAGT	3731
Hypercholesterolaemia Asp203Gly GAC-GGC	GCCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGAT GGTGGCCCCGACTGCAAGGACAAATCTGACGAGGAAAACTG CGGTATGGGCGGGGCCAGGGTGGGGGCGGGGCGTCCTAT	3732
	ATAGGACGCCCCGCCCCCACCCTGGCCCCGCCCATACCGCA GTTTTCCTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCATC ACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGC	3733
	CTGCAAGGACAAATCTG	3734
	CAGATTTGTCCTTGCAG	3735
Hypercholesterolaemia Asp203Val GAC-GTC	GCCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGAT GGTGGCCCCGACTGCAAGGACAAATCTGACGAGGAAAACTG CGGTATGGGCGGGGCCAGGGTGGGGGCGGGGCGTCCTAT	3736
	ATAGGACGCCCCGCCCCCACCCTGGCCCCGCCCATACCGCA GTTTTCCTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCATC ACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGC	3737
	CTGCAAGGACAAATCTG	3738
	CAGATTTGTCCTTGCAG	3739
Hypercholesterolaemia Ser205Pro aTCT-CCT	AGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGATGGTGG CCCCGACTGCAAGGACAAATCTGACGAGGAAAACTGCGGTAT GGGCGGGGCCAGGGTGGGGGCGGGGCGTCCTATCACCT	3740
	AGGTGATAGGACGCCCCGCCCCCACCCTGGCCCCGCCCATA CCGCAGTTTTCCTCGTCAGATTTGTCCTTGCAGTCGGGGCCA CCATCACAGCGCCAGCTGGAGTGGATGCACTCGCCACT	3741
	AGGACAAATCTGACGAG	3742
	CTCGTCAGATTTGTCCT	3743
Hypercholesterolaemia Asp206Glu GACg-GAG	CGAGTGCATCCACTCCAGCTGGCGCTGTGATGGTGGCCCCG ACTGCAAGGACAAATCTGACGAGGAAAACTGCGGTATGGGC GGGGCCAGGGTGGGGGCGGGGCGTCCTATCACCTGTCCC	3744
	GGGACAGGTGATAGGACGCCCCGCCCCCACCCTGGCCCCG CCCATACCGCAGTTTTCCTCGTCAGATTTGTCCTTGCAGTCG GGGCCACCATCACAGCGCCAGCTGGAGTGGATGCACTCG	3745
	AAATCTGACGAGGAAAA	3746
	TTTTCCTCGTCAGATTT	3747
Hypercholesterolaemia Glu207Gln cGAG-CAG	GAGTGCATCCACTCCAGCTGGCGCTGTGATGGTGGCCCCGA CTGCAAGGACAAATCTGACGAGGAAAACTGCGGTATGGGCG GGGCCAGGGTGGGGGCGGGGCGTCCTATCACCTGTCCCT	3748
	AGGGACAGGTGATAGGACGCCCCGCCCCCACCCTGGCCCC GCCCATACCGCAGTTTTCCTCGTCAGATTTGTCCTTGCAGTC GGGGCCACCATCACAGCGCCAGCTGGAGTGGATGCACTC	3749
	AATCTGACGAGGAAAAC	3750
	GTTTTCCTCGTCAGATT	3751

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Hypercholesterolaemia Glu207Lys cGAG-AAG	GAGTGCATCCACTCCAGCTGGCGCTGTGATGGTGGCCCCGA CTGCAAGGACAAATCTGAC**G**AGGAAAACTGCGGTATGGGCG GGGCCAGGGTGGGGGCGGGGCGTCCTATCACCTGTCCCT	3752
	AGGGACAGGTGATAGGACGCCCCGCCCCCACCCTGGCCCC GCCCATACCGCAGTTTTCCT**C**GTCAGATTTGTCCTTGCAGTC GGGGCCACCATCACAGCGCCAGCTGGAGTGGATGCACTC	3753
	AATCTGAC**G**AGGAAAAC	3754
	GTTTTCCT**C**GTCAGATT	3755
Hypercholesterolaemia Glu207Term cGAG-TAG	GAGTGCATCCACTCCAGCTGGCGCTGTGATGGTGGCCCCGA CTGCAAGGACAAATCTGAC**G**AGGAAAACTGCGGTATGGGCG GGGCCAGGGTGGGGGCGGGGCGTCCTATCACCTGTCCCT	3756
	AGGGACAGGTGATAGGACGCCCCGCCCCCACCCTGGCCCC GCCCATACCGCAGTTTTCCT**C**GTCAGATTTGTCCTTGCAGTC GGGGCCACCATCACAGCGCCAGCTGGAGTGGATGCACTC	3757
	AATCTGAC**G**AGGAAAAC	3758
	GTTTTCCT**C**GTCAGATT	3759
Hypercholesterolaemia Glu219Lys cGAA-AAA	TCTTGAGAAAATCAACACACTCTGTCCTGTTTTCCAGCTGTGG CCACCTGTCGCCCTGAC**G**AATTCCAGTGCTCTGATGGAAACT GCATCCATGGCAGCCGGCAGTGTGACCGGGAATATG	3760
	CATATTCCCGGTCACACTGCCGGCTGCCATGGATGCAGTTTC CATCAGAGCACTGGAATT**C**GTCAGGGCGACAGGTGGCCACA GCTGGAAAACAGGACAGAGTGTGTTGATTTTCTCAAGA	3761
	GCCCTGAC**G**AATTCCAG	3762
	CTGGAATT**C**GTCAGGGC	3763
Hypercholesterolaemia Gln221Term cCAG-TAG	GAAAATCAACACACTCTGTCCTGTTTTCCAGCTGTGGCCACCT GTCGCCCTGACGAATTC**C**AGTGCTCTGATGGAAACTGCATCC ATGGCAGCCGGCAGTGTGACCGGGAATATGACTGCA	3764
	TGCAGTCATATTCCCGGTCACACTGCCGGCTGCCATGGATGC AGTTTCCATCAGAGCACT**G**GAATTCGTCAGGGCGACAGGTGG CCACAGCTGGAAAACAGGACAGAGTGTGTTGATTTTC	3765
	ACGAATTC**C**AGTGCTCT	3766
	AGAGCACT**G**GAATTCGT	3767
Hypercholesterolaemia Cys227Phe TGC-TTC	CCTGTTTTCCAGCTGTGGCCACCTGTCGCCCTGACGAATTCC AGTGCTCTGATGGAAACT**G**CATCCATGGCAGCCGGCAGTGT GACCGGGAATATGACTGCAAGGACATGAGCGATGAAGT	3768
	ACTTCATCGCTCATGTCCTTGCAGTCATATTCCCGGTCACACT GCCGGCTGCCATGGATG**C**AGTTTCCATCAGAGCACTGGAATT CGTCAGGGCGACAGGTGGCCACAGCTGGAAAACAGG	3769
	TGGAAACT**G**CATCCATG	3770
	CATGGATG**C**AGTTTCCA	3771
Hypercholesterolaemia Asp235Glu GACc-GAA	T[illegible]CCCTGACGAATTCCAGTGCTCTGATGGAAACTGCATCCA TGGCAGCCGGCAGTGTGA**C**CGGGAATATGACTGCAAGGACA TGAGCGATGAAGTTGGCTGCGTTAATGGTGAGCGCTGG	3772

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCAGCGCTCACCATTAACGCAGCCAACTTCATCGCTCATGTC CTTGCAGTCATATTCCCGGTCACACTGCCGGCTGCCATGGAT GCAGTTTCCATCAGAGCACTGGAATTCGTCAGGGCGA	3773
	CAGTGTGACCGGGAATA	3774
	TATTCCCGGTCACACTG	3775
Hypercholesterolaemia Asp235Gly GAC-GGC	GTCGCCCTGACGAATTCCAGTGCTCTGATGGAAACTGCATCC ATGGCAGCCGGCAGTGTGACCGGGAATATGACTGCAAGGAC ATGAGCGATGAAGTTGGCTGCGTTAATGGTGAGCGCTG	3776
	CAGCGCTCACCATTAACGCAGCCAACTTCATCGCTCATGTCC TTGCAGTCATATTCCCGGTCACACTGCCGGCTGCCATGGATG CAGTTTCCATCAGAGCACTGGAATTCGTCAGGGCGAC	3777
	GCAGTGTGACCGGGAAT	3778
	ATTCCCGGTCACACTGC	3779
Hypercholesterolaemia Glu237Lys gGAA-AAA	CCTGACGAATTCCAGTGCTCTGATGGAAACTGCATCCATGGC AGCCGGCAGTGTGACCGGGAATATGACTGCAAGGACATGAG CGATGAAGTTGGCTGCGTTAATGGTGAGCGCTGGCCAT	3780
	ATGGCCAGCGCTCACCATTAACGCAGCCAACTTCATCGCTCA TGTCCTTGCAGTCATATTCCCGGTCACACTGCCGGCTGCCAT GGATGCAGTTTCCATCAGAGCACTGGAATTCGTCAGG	3781
	GTGACCGGGAATATGAC	3782
	GTCATATTCCCGGTCAC	3783
Hypercholesterolaemia Cys240Phe TGC-TTC	TCCAGTGCTCTGATGGAAACTGCATCCATGGCAGCCGGCAGT GTGACCGGGAATATGACTGCAAGGACATGAGCGATGAAGTTG GCTGCGTTAATGGTGAGCGCTGGCCATCTGGTTTTCC	3784
	GGAAAACCAGATGGCCAGCGCTCACCATTAACGCAGCCAACT TCATCGCTCATGTCCTTGCAGTCATATTCCCGGTCACACTGC CGGCTGCCATGGATGCAGTTTCCATCAGAGCACTGGA	3785
	ATATGACTGCAAGGACA	3786
	TGTCCTTGCAGTCATAT	3787
Hypercholesterolaemia Asp245Glu GATg-GAA	AAACTGCATCCATGGCAGCCGGCAGTGTGACCGGGAATATG ACTGCAAGGACATGAGCGATGAAGTTGGCTGCGTTAATGGTG AGCGCTGGCCATCTGGTTTTCCATCCCCCATTCTCTGT	3788
	ACAGAGAATGGGGGATGGAAAACCAGATGGCCAGCGCTCAC CATTAACGCAGCCAACTTCATCGCTCATGTCCTTGCAGTCATA TTCCCGGTCACACTGCCGGCTGCCATGGATGCAGTTT	3789
	ATGAGCGATGAAGTTGG	3790
	CCAACTTCATCGCTCAT	3791
Hypercholesterolaemia Cys249Tyr TGC-TAC	ATGGCAGCCGGCAGTGTGACCGGGAATATGACTGCAAGGAC ATGAGCGATGAAGTTGGCTGCGTTAATGGTGAGCGCTGGCC ATCTGGTTTTCCATCCCCCATTCTCTGTGCCTTGCTGCT	3792
	AGCAGCAAGGCACAGAGAATGGGGGATGGAAAACCAGATGG CCAGCGCTCACCATTAACGCAGCCAACTTCATCGCTCATGTC CTTGCAGTCATATTCCCGGTCACACTGCCGGCTGCCAT	3793
	AGTTGGCTGCGTTAATG	3794

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CATTAACGCAGCCAACT	3795
Hypercholesterolaemia Glu256Lys cGAG-AAG	AGGCTCAGACACACCTGACCTTCCTCCTTCCTCTCTCTGGCT CTCACAGTGACACTCTGCGAGGGACCCAACAAGTTCAAGTGT CACAGCGGCGAATGCATCACCCTGGACAAAGTCTGCA	3796
	TGCAGACTTTGTCCAGGGTGATGCATTCGCCGCTGTGACACT TGAACTTGTTGGGTCCCTCGCAGAGTGTCACTGTGAGAGCCA GAGAGAGGAAGGAGGAAGGTCAGGTGTGTCTGAGCCT	3797
	CACTCTGCGAGGGACCC	3798
	GGGTCCCTCGCAGAGTG	3799
Hypercholesterolaemia Ser265Arg AGCg-AGA	CCTCTCTCTGGCTCTCACAGTGACACTCTGCGAGGGACCCAA CAAGTTCAAGTGTCACAGCGGCGAATGCATCACCCTGGACAA AGTCTGCAACATGGCTAGAGACTGCCGGGACTGGTCA	3800
	TGACCAGTCCCGGCAGTCTCTAGCCATGTTGCAGACTTTGTC CAGGGTGATGCATTCGCCGCTGTGACACTTGAACTTGTTGGG TCCCTCGCAGAGTGTCACTGTGAGAGCCAGAGAGAGG	3801
	TGTCACAGCGGCGAATG	3802
	CATTCGCCGCTGTGACA	3803
Hypercholesterolaemia Glu267Lys cGAA-AAA	TCTCTGGCTCTCACAGTGACACTCTGCGAGGGACCCAACAAG TTCAAGTGTCACAGCGGCGAATGCATCACCCTGGACAAAGTC TGCAACATGGCTAGAGACTGCCGGGACTGGTCAGATG	3804
	CATCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGCAGACTT TGTCCAGGGTGATGCATTCGCCGCTGTGACACTTGAACTTGT TGGGTCCCTCGCAGAGTGTCACTGTGAGAGCCAGAGA	3805
	ACAGCGGCGAATGCATC	3806
	GATGCATTCGCCGCTGT	3807
Hypercholesterolaemia Glu267Term cGAA-TAA	TCTCTGGCTCTCACAGTGACACTCTGCGAGGGACCCAACAAG TTCAAGTGTCACAGCGGCGAATGCATCACCCTGGACAAAGTC TGCAACATGGCTAGAGACTGCCGGGACTGGTCAGATG	3808
	CATCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGCAGACTT TGTCCAGGGTGATGCATTCGCCGCTGTGACACTTGAACTTGT TGGGTCCCTCGCAGAGTGTCACTGTGAGAGCCAGAGA	3809
	ACAGCGGCGAATGCATC	3810
	GATGCATTCGCCGCTGT	3811
Hypercholesterolaemia Lys273Glu cAAA-GAA	ACACTCTGCGAGGGACCCAACAAGTTCAAGTGTCACAGCGG CGAATGCATCACCCTGGACAAAGTCTGCAACATGGCTAGAGA CTGCCGGGACTGGTCAGATGAACCCATCAAAGAGTGCG	3812
	CGCACTCTTTGATGGGTTCATCTGACCAGTCCCGGCAGTCTC TAGCCATGTTGCAGACTTTGTCCAGGGTGATGCATTCGCCGC TGTGACACTTGAACTTGTTGGGTCCCTCGCAGAGTGT	3813
	CCCTGGACAAAGTCTGC	3814
	GCAGACTTTGTCCAGGG	3815
Hypercholesterolaemia Cys275Term TGCa-TGA	CGAGGGACCCAACAAGTTCAAGTGTCACAGCGGCGAATGCA TCACCCTGGACAAAGTCTGCAACATGGCTAGAGACTGCCGG GACTGGTCAGATGAACCCATCAAAGAGTGCGGTGAGTCT	3816

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGACTCACCGCACTCTTTGATGGGTTCATCTGACCAGTCCCG GCAGTCTCTAGCCATGTTGCAGACTTTGTCCAGGGTGATGCA TTCGCCGCTGTGACACTTGAACTTGTTGGGTCCCTCG	3817
	AAAGTCTGCAACATGGC	3818
	GCCATGTTGCAGACTTT	3819
Hypercholesterolaemia Asp280Gly GAC-GGC	AGTTCAAGTGTCACAGCGGCGAATGCATCACCCTGGACAAAG TCTGCAACATGGCTAGAGACTGCCGGGACTGGTCAGATGAA CCCATCAAAGAGTGCGGTGAGTCTCGGTGCAGGCGGCT	3820
	AGCCGCCTGCACCGAGACTCACCGCACTCTTTGATGGGTTCA TCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGCAGACTTTG TCCAGGGTGATGCATTCGCCGCTGTGACACTTGAACT	3821
	GGCTAGAGACTGCCGGG	3822
	CCCGGCAGTCTCTAGCC	3823
Hypercholesterolaemia Cys281Tyr TGC-TAC	TCAAGTGTCACAGCGGCGAATGCATCACCCTGGACAAAGTCT GCAACATGGCTAGAGACTGCCGGGACTGGTCAGATGAACCC ATCAAAGAGTGCGGTGAGTCTCGGTGCAGGCGGCTTGC	3824
	GCAAGCCGCCTGCACCGAGACTCACCGCACTCTTTGATGGG TTCATCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGCAGAC TTTGTCCAGGGTGATGCATTCGCCGCTGTGACACTTGA	3825
	TAGAGACTGCCGGGACT	3826
	AGTCCCGGCAGTCTCTA	3827
Hypercholesterolaemia Asp283Asn gGAC-AAC	TGTCACAGCGGCGAATGCATCACCCTGGACAAAGTCTGCAAC ATGGCTAGAGACTGCCGGGACTGGTCAGATGAACCCATCAAA GAGTGCGGTGAGTCTCGGTGCAGGCGGCTTGCAGAGT	3828
	ACTCTGCAAGCCGCCTGCACCGAGACTCACCGCACTCTTTGA TGGGTTCATCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGC AGACTTTGTCCAGGGTGATGCATTCGCCGCTGTGACA	3829
	ACTGCCGGGACTGGTCA	3830
	TGACCAGTCCCGGCAGT	3831
Hypercholesterolaemia Asp283Glu GACt-GAG	TCACAGCGGCGAATGCATCACCCTGGACAAAGTCTGCAACAT GGCTAGAGACTGCCGGGACTGGTCAGATGAACCCATCAAAG AGTGCGGTGAGTCTCGGTGCAGGCGGCTTGCAGAGTTT	3832
	AAACTCTGCAAGCCGCCTGCACCGAGACTCACCGCACTCTTT GATGGGTTCATCTGACCAGTCCCGGCAGTCTCTAGCCATGTT GCAGACTTTGTCCAGGGTGATGCATTCGCCGCTGTGA	3833
	TGCCGGGACTGGTCAGA	3834
	TCTGACCAGTCCCGGCA	3835
Hypercholesterolaemia Asp283Tyr gGAC-TAC	TGTCACAGCGGCGAATGCATCACCCTGGACAAAGTCTGCAAC ATGGCTAGAGACTGCCGGGACTGGTCAGATGAACCCATCAAA GAGTGCGGTGAGTCTCGGTGCAGGCGGCTTGCAGAGT	3836
	ACTCTGCAAGCCGCCTGCACCGAGACTCACCGCACTCTTTGA TGGGTTCATCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGC AGACTTTGTCCAGGGTGATGCATTCGCCGCTGTGACA	3837
	ACTGCCGGGACTGGTCA	3838

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGACCAGTCCCGGCAGT	3839
Hypercholesterolaemia Trp284Term TGGt-TGA	CAGCGGCGAATGCATCACCCTGGACAAAGTCTGCAACATGG CTAGAGACTGCCGGGACTGGTCAGATGAACCCATCAAAGAGT GCGGTGAGTCTCGGTGCAGGCGGCTTGCAGAGTTTGTG	3840
	CACAAACTCTGCAAGCCGCCTGCACCGAGACTCACCGCACT CTTTGATGGGTTCATCTGACCAGTCCCGGCAGTCTCTAGCCA TGTTGCAGACTTTGTCCAGGGTGATGCATTCGCCGCTG	3841
	CGGGACTGGGTCAGATGA	3842
	TCATCTGACCAGTCCCG	3843
Hypercholesterolaemia Ser285Leu TCA-TTA	GCGGCGAATGCATCACCCTGGACAAAGTCTGCAACATGGCTA GAGACTGCCGGGACTGGTCAGATGAACCCATCAAAGAGTGC GGTGAGTCTCGGTGCAGGCGGCTTGCAGAGTTTGTGGG	3844
	CCCACAAACTCTGCAAGCCGCCTGCACCGAGACTCACCGCA CTCTTTGATGGGTTCATCTGACCAGTCCCGGCAGTCTCTAGC CATGTTGCAGACTTTGTCCAGGGTGATGCATTCGCCGC	3845
	GGACTGGTCAGATGAAC	3846
	GTTCATCTGACCAGTCC	3847
Hypercholesterolaemia Lys290Arg AAA-AGA	CCCTGGACAAAGTCTGCAACATGGCTAGAGACTGCCGGGAC TGGTCAGATGAACCCATCAAAGAGTGCGGTGAGTCTCGGTG CAGGCGGCTTGCAGAGTTTGTGGGGAGCCAGGAAAGGGA	3848
	TCCCTTTCCTGGCTCCCCACAAACTCTGCAAGCCGCCTGCAC CGAGACTCACCGCACTCTTTGATGGGTTCATCTGACCAGTCC CGGCAGTCTCTAGCCATGTTGCAGACTTTGTCCAGGG	3849
	ACCCATCAAAGAGTGCG	3850
	CGCACTCTTTGATGGGT	3851
Hypercholesterolaemia Cys297Phe TGC-TTC	GGGTAGGGGCCCGAGAGTGACCAGTCTGCATCCCCTGGCCC TGCGCAGGGACCAACGAATGCTTGGACAACAACGGCGGCTG TTCCCACGTCTGCAATGACCTTAAGATCGGCTACGAGTG	3852
	CACTCGTAGCCGATCTTAAGGTCATTGCAGACGTGGGAACAG CCGCCGTTGTTGTCCAAGCATTCGTTGGTCCCTGCGCAGGG CCAGGGGATGCAGACTGGTCACTCTCGGGCCCCTACCC	3853
	CAACGAATGCTTGGACA	3854
	TGTCCAAGCATTCGTTG	3855
Hypercholesterolaemia Cys297Tyr TGC-TAC	GGGTAGGGGCCCGAGAGTGACCAGTCTGCATCCCCTGGCCC TGCGCAGGGACCAACGAATGCTTGGACAACAACGGCGGCTG TTCCCACGTCTGCAATGACCTTAAGATCGGCTACGAGTG	3856
	CACTCGTAGCCGATCTTAAGGTCATTGCAGACGTGGGAACAG CCGCCGTTGTTGTCCAAGCATTCGTTGGTCCCTGCGCAGGG CCAGGGGATGCAGACTGGTCACTCTCGGGCCCCTACCC	3857
	CAACGAATGCTTGGACA	3858
	TGTCCAAGCATTCGTTG	3859
Hypercholesterolaemia His306Tyr cCAC-TAC	TGCATCCCCTGGCCCTGCGCAGGGACCAACGAATGCTTGGA CAACAACGGCGGCTGTTCCCACGTCTGCAATGACCTTAAGAT CGGCTACGAGTGCCTGTGCCCCGACGGCTTCCAGCTGG	3860

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTAGCCGATC TTAAGGTCATTGCAGACGTGGGAACAGCCGCCGTTGTTGTCC AAGCATTCGTTGGTCCCTGCGCAGGGCCAGGGGATGCA	3861
	GCTGTTCCCACGTCTGC	3862
	GCAGACGTGGGAACAGC	3863
Hypercholesterolaemia Cys308Gly cTGC-GGC	CCCTGGCCCTGCGCAGGGACCAACGAATGCTTGGACAACAA CGGCGGCTGTTCCCACGTCTTGCAATGACCTTAAGATCGGCTA CGAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCCC	3864
	GGGCCACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTA GCCGATCTTAAGGTCATTGCAGACGTGGGAACAGCCGCCGT TGTTGTCCAAGCATTCGTTGGTCCCTGCGCAGGGCCAGGG	3865
	CCCACGTCTTGCAATGAC	3866
	GTCATTGCAGACGTGGG	3867
Hypercholesterolaemia Cys308Tyr TGC-TAC	CCTGGCCCTGCGCAGGGACCAACGAATGCTTGGACAACAAC GGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCGGCTAC GAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCCCA	3868
	TGGGCCACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTA GCCGATCTTAAGGTCATTGCAGACGTGGGAACAGCCGCCGTT GTTGTCCAAGCATTCGTTGGTCCCTGCGCAGGGCCAGG	3869
	CCACGTCTGCAATGACC	3870
	GGTCATTGCAGACGTGG	3871
Hypercholesterolaemia Gly314Ser cGGC-AGC	ACCAACGAATGCTTGGACAACAACGGCGGCTGTTCCCACGTC TGCAATGACCTTAAGATCGGCTACGAGTGCCTGTGCCCCGAC GGCTTCCAGCTGGTGGCCCAGCGAAGATGCGAAGGTG	3872
	CACCTTCGCATCTTCGCTGGGCCACCAGCTGGAAGCCGTCG GGGCACAGGCACTCGTAGCCGATCTTAAGGTCATTGCAGAC GTGGGAACAGCCGCCGTTGTTGTCCAAGCATTCGTTGGT	3873
	TTAAGATCGGCTACGAG	3874
	CTCGTAGCCGATCTTAA	3875
Hypercholesterolaemia Gly314Val GGC-GTC	CCAACGAATGCTTGGACAACAACGGCGGCTGTTCCCACGTCT GCAATGACCTTAAGATCGGCTACGAGTGCCTGTGCCCCGAC GGCTTCCAGCTGGTGGCCCAGCGAAGATGCGAAGGTGA	3876
	TCACCTTCGCATCTTCGCTGGGCCACCAGCTGGAAGCCGTC GGGGCACAGGCACTCGTAGCCGATCTTAAGGTCATTGCAGA CGTGGGAACAGCCGCCGTTGTTGTCCAAGCATTCGTTGG	3877
	TAAGATCGGCTACGAGT	3878
	ACTCGTAGCCGATCTTA	3879
Hypercholesterolaemia Tyr315Term TACg-TAA	CGAATGCTTGGACAACAACGGCGGCTGTTCCCACGTCTGCAA TGACCTTAAGATCGGCTACGAGTGCCTGTGCCCCGACGGCTT CCAGCTGGTGGCCCAGCGAAGATGCGAAGGTGATTTC	3880
	GAAATCACCTTCGCATCTTCGCTGGGCCACCAGCTGGAAGCC CCGGGGCACAGGCACTCGTAGCCGATCTTAAGGTCATTGCA GACGTGGGAACAGCCGCCGTTGTTGTCCAAGCATTCG	3881
	ATCGGCTACGAGTGCCT	3882

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGGCACTCGTAGCCGAT	3883
Hypercholesterolaemia Cys317Gly gTGC-GGC	TGCTTGGACAACAACGGCGGCTGTTCCCACGTCTGCAATGAC CTTAAGATCGGCTACGAGTGCCTGTGCCCCGACGGCTTCCA GCTGGTGGCCCAGCGAAGATGCGAAGGTGATTTCCGGG	3884
	CCCGGAAATCACCTTCGCATCTTCGCTGGGCCACCAGCTGG AAGCCGTCGGGGCACAGGCACTCGTAGCCGATCTTAAGGTC ATTGCAGACGTGGGAACAGCCGCCGTTGTTGTCCAAGCA	3885
	GCTACGAGTGCCTGTGC	3886
	GCACAGGCACTCGTAGC	3887
Hypercholesterolaemia Cys317Ser gTGC-AGC	TGCTTGGACAACAACGGCGGCTGTTCCCACGTCTGCAATGAC CTTAAGATCGGCTACGAGTGCCTGTGCCCCGACGGCTTCCA GCTGGTGGCCCAGCGAAGATGCGAAGGTGATTTCCGGG	3888
	CCCGGAAATCACCTTCGCATCTTCGCTGGGCCACCAGCTGG AAGCCGTCGGGGCACAGGCACTCGTAGCCGATCTTAAGGTC ATTGCAGACGTGGGAACAGCCGCCGTTGTTGTCCAAGCA	3889
	GCTACGAGTGCCTGTGC	3890
	GCACAGGCACTCGTAGC	3891
Hypercholesterolaemia Pro320Arg CCC-CGC	ACAACGGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCG GCTACGAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCC CAGCGAAGATGCGAAGGTGATTTCCGGGTGGGACTGAG	3892
	CTCAGTCCCACCCGGAAATCACCTTCGCATCTTCGCTGGGCC ACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTAGCCGAT CTTAAGGTCATTGCAGACGTGGGAACAGCCGCCGTTGT	3893
	CCTGTGCCCCGACGGCT	3894
	AGCCGTCGGGGCACAGG	3895
Hypercholesterolaemia Asp321Asn cGAC-AAC	AACGGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCGGC TACGAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCCCA GCGAAGATGCGAAGGTGATTTCCGGGTGGGACTGAGCC	3896
	GGCTCAGTCCCACCCGGAAATCACCTTCGCATCTTCGCTGGG CCACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTAGCCG ATCTTAAGGTCATTGCAGACGTGGGAACAGCCGCCGTT	3897
	TGTGCCCCGACGGCTTC	3898
	GAAGCCGTCGGGGCACA	3899
Hypercholesterolaemia Asp321Glu GACg-GAG	CGGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCGGCTA CGAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCCCAGC GAAGATGCGAAGGTGATTTCCGGGTGGGACTGAGCCCT	3900
	AGGGCTCAGTCCCACCCGGAAATCACCTTCGCATCTTCGCTG GGCCACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTAG CCGATCTTAAGGTCATTGCAGACGTGGGAACAGCCGCCG	3901
	TGCCCCGACGGCTTCCA	3902
	TGGAAGCCGTCGGGGCA	3903
Hypercholesterolaemia Gly322Ser cGGC-AGC	GGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCGGCTAC GAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCCCAGCG AAGATGCGAAGGTGATTTCCGGGTGGGACTGAGCCCTG	3904

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CAGGGCTCAGTCCCACCCGGAAATCACCTTCGCATCTTCGCT GGGCCACCAGCTGGAAGC**C**GTCGGGGCACAGGCACTCGTA GCCGATCTTAAGGTCATTGCAGACGTGGGAACAGCCGCC	3905
	GCCCCGAC**G**GCTTCCAG	3906
	CTGGAAGC**C**GTCGGGGC	3907
Hypercholesterolaemia Gln324Term cCAG-TAG	TGTTCCCACGTCTGCAATGACCTTAAGATCGGCTACGAGTGC CTGTGCCCCGACGGCTTC**C**AGCTGGTGGCCCAGCGAAGATG CGAAGGTGATTTCCGGGTGGGACTGAGCCCTGGGCCCC	3908
	GGGGCCCAGGGCTCAGTCCCACCCGGAAATCACCTTCGCAT CTTCGCTGGGCCACCAGCT**G**GAAGCCGTCGGGGCACAGGCA CTCGTAGCCGATCTTAAGGTCATTGCAGACGTGGGAACA	3909
	ACGGCTTC**C**AGCTGGTG	3910
	CACCAGCT**G**GAAGCCGT	3911
Hypercholesterolaemia Arg329Pro CGA-CCA	ATGACCTTAAGATCGGCTACGAGTGCCTGTGCCCCGACGGC TTCCAGCTGGTGGCCCAGC**G**AAGATGCGAAGGTGATTTCCG GGTGGGACTGAGCCCTGGGCCCCCTCTGCGCTTCCTGAC	3912
	GTCAGGAAGCGCAGAGGGGGCCCAGGGCTCAGTCCCACCC GGAAATCACCTTCGCATCTT**C**GCTGGGCCACCAGCTGGAAG CCGTCGGGGCACAGGCACTCGTAGCCGATCTTAAGGTCAT	3913
	GGCCCAGC**G**AAGATGCG	3914
	CGCATCTT**C**GCTGGGCC	3915
Hypercholesterolaemia Arg329Term gCGA-TGA	AATGACCTTAAGATCGGCTACGAGTGCCTGTGCCCCGACGG CTTCCAGCTGGTGGCCCAG**C**GAAGATGCGAAGGTGATTTCC GGGTGGGACTGAGCCCTGGGCCCCCTCTGCGCTTCCTGA	3916
	TCAGGAAGCGCAGAGGGGGCCCAGGGCTCAGTCCCACCCG GAAATCACCTTCGCATCTTC**G**CTGGGCCACCAGCTGGAAGCC GTCGGGGCACAGGCACTCGTAGCCGATCTTAAGGTCATT	3917
	TGGCCCAG**C**GAAGATGC	3918
	GCATCTTC**G**CTGGGCCA	3919
Hypercholesterolaemia Glu336Lys tGAG-AAG	TCTAGCCATTGGGGAAGAGCCTCCCCACCAAGCCTCTTTCTC TCTCTTCCAGATATCGAT**G**AGTGTCAGGATCCCGACACCTGC AGCCAGCTCTGCGTGAACCTGGAGGGTGGCTACAAGT	3920
	ACTTGTAGCCACCCTCCAGGTTCACGCAGAGCTGGCTGCAG GTGTCGGGATCCTGACACT**C**ATCGATATCTGGAAGAGAGAGA AAGAGGCTTGGTGGGGAGGCTCTTCCCCAATGGCTAGA	3921
	ATATCGAT**G**AGTGTCAG	3922
	CTGACACT**C**ATCGATAT	3923
Hypercholesterolaemia Gln338Term tCAG-TAG	CATTGGGGAAGAGCCTCCCCACCAAGCCTCTTTCTCTCTCTT CCAGATATCGATGAGTGT**C**AGGATCCCGACACCTGCAGCCAG CTCTGCGTGAACCTGGAGGGTGGCTACAAGTGCCAGT	3924
	ACTGGCACTTGTAGCCACCCTCCAGGTTCACGCAGAGCTGG CTGCAGGTGTCGGGATCCT**G**ACACTCATCGATATC, GGAAGA GAGAGAAAGAGGCTTGGTGGGGAGGCTCTTCCCCAATG	3925
	ATGAGTGT**C**AGGATCCC	3926

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GGGATCCTGACACTCAT	3927
Hypercholesterolaemia Cys343Arg cTGC-CGC	TCCCCACCAAGCCTCTTTCTCTCTCTTCCAGATATCGATGAGT GTCAGGATCCCGACACCTGCAGCCAGCTCTGCGTGAACCTG GAGGGTGGCTACAAGTGCCAGTGTGAGGAAGGCTTCC	3928
	GGAAGCCTTCCTCACACTGGCACTTGTAGCCACCCTCCAGGT TCACGCAGAGCTGGCTGCAGGTGTCGGGATCCTGACACTCA TCGATATCTGGAAGAGAGAGAAAGAGGCTTGGTGGGGA	3929
	CCGACACCTGCAGCCAG	3930
	CTGGCTGCAGGTGTCGG	3931
Hypercholesterolaemia Gln345Arg CAG-CGG	CAAGCCTCTTTCTCTCTCTTCCAGATATCGATGAGTGTCAGGA TCCCGACACCTGCAGCCAGCTCTGCGTGAACCTGGAGGGTG GCTACAAGTGCCAGTGTGAGGAAGGCTTCCAGCTGGA	3932
	TCCAGCTGGAAGCCTTCCTCACACTGGCACTTGTAGCCACCC TCCAGGTTCACGCAGAGCTGGCTGCAGGTGTCGGGATCCTG ACACTCATCGATATCTGGAAGAGAGAGAAAGAGGCTTG	3933
	CTGCAGCCAGCTCTGCG	3934
	CGCAGAGCTGGCTGCAG	3935
Hypercholesterolaemia Cys347Tyr TGC-TAC	TCTTTCTCTCTCTTCCAGATATCGATGAGTGTCAGGATCCCGA CACCTGCAGCCAGCTCTGCGTGAACCTGGAGGGTGGCTACA AGTGCCAGTGTGAGGAAGGCTTCCAGCTGGACCCCCA	3936
	TGGGGGTCCAGCTGGAAGCCTTCCTCACACTGGCACTTGTA GCCACCCTCCAGGTTCACGCAGAGCTGGCTGCAGGTGTCGG GATCCTGACACTCATCGATATCTGGAAGAGAGAGAAAGA	3937
	CCAGCTCTGCGTGAACC	3938
	GGTTCACGCAGAGCTGG	3939
Hypercholesterolaemia Cys347Arg cTGC-CGC	CTCTTTCTCTCTCTTCCAGATATCGATGAGTGTCAGGATCCCG ACACCTGCAGCCAGCTCTGCGTGAACCTGGAGGGTGGCTAC AAGTGCCAGTGTGAGGAAGGCTTCCAGCTGGACCCCC	3940
	GGGGGTCCAGCTGGAAGCCTTCCTCACACTGGCACTTGTAG CCACCCTCCAGGTTCACGCAGAGCTGGCTGCAGGTGTCGGG ATCCTGACACTCATCGATATCTGGAAGAGAGAGAAAGAG	3941
	GCCAGCTCTGCGTGAAC	3942
	GTTCACGCAGAGCTGGC	3943
Hypercholesterolaemia Gly352Asp GGT-GAT	CAGATATCGATGAGTGTCAGGATCCCGACACCTGCAGCCAGC TCTGCGTGAACCTGGAGGGTGGCTACAAGTGCCAGTGTGAG GAAGGCTTCCAGCTGGACCCCCACACGAAGGCCTGCAA	3944
	TTGCAGGCCTTCGTGTGGGGGTCCAGCTGGAAGCCTTCCTC ACACTGGCACTTGTAGCCACCCTCCAGGTTCACGCAGAGCTG GCTGCAGGTGTCGGGATCCTGACACTCATCGATATCTG	3945
	CCTGGAGGGTGGCTACA	3946
	TGTAGCCACCCTCCAGG	3947
Hypercholesterolaemia Tyr354Cys TAC-TGC	TCGATGAGTGTCAGGATCCCGACACCTGCAGCCAGCTCTGC GTGAACCTGGAGGGTGGCTACAAGTGCCAGTGTGAGGAAGG CTTCCAGCTGGACCCCCACACGAAGGCCTGCAAGGCTGT	3948

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACAGCCTTGCAGGCCTTCGTGTGGGGGTCCAGCTGGAAGCC TTCCTCACACTGGCACTTGTAGCCACCCTCCAGGTTCACGCA GAGCTGGCTGCAGGTGTCGGGATCCTGACACTCATCGA	3949
	GGGTGGCTACAAGTGCC	3950
	GGCACTTGTAGCCACCC	3951
Hypercholesterolaemia Cys358Arg gTGT-CGT	CAGGATCCCGACACCTGCAGCCAGCTCTGCGTGAACCTGGA GGGTGGCTACAAGTGCCAGTGTGAGGAAGGCTTCCAGCTGG ACCCCCACACGAAGGCCTGCAAGGCTGTGGGTGAGCACG	3952
	CGTGCTCACCCACAGCCTTGCAGGCCTTCGTGTGGGGGTCC AGCTGGAAGCCTTCCTCACACTGGCACTTGTAGCCACCCTCC AGGTTCACGCAGAGCTGGCTGCAGGTGTCGGGATCCTG	3953
	AGTGCCAGTGTGAGGAA	3954
	TTCCTCACACTGGCACT	3955
Hypercholesterolaemia Gln363Term cCAG-TAG	TGCAGCCAGCTCTGCGTGAACCTGGAGGGTGGCTACAAGTG CCAGTGTGAGGAAGGCTTCCAGCTGGACCCCCACACGAAGG CCTGCAAGGCTGTGGGTGAGCACGGGAAGGCGGCGGGTG	3956
	CACCCGCCGCCTTCCCGTGCTCACCCACAGCCTTGCAGGCC TTCGTGTGGGGGTCCAGCTGGAAGCCTTCCTCACACTGGCA CTTGTAGCCACCCTCCAGGTTCACGCAGAGCTGGCTGCA	3957
	AAGGCTTCCAGCTGGAC	3958
	GTCCAGCTGGAAGCCTT	3959

EXAMPLE 22
UDP-glucuronosyltransferase - UGT1

Mutations in the human UGT1 gene result in a range of disease syndromes, ranging from relatively common diseases such as Gilbert's syndrome, which effects up to 7% of the population, to rare disorders such as Crigler-Najjar syndrome. Symptoms of these diseases are the result of diminished bilirubin conjugation and typically present with jaundice or, when mild, as an incidental finding during routing laboratory analysis. Severe cases of Crigler-Najjar syndrome are caused by an absence of UGT1 activity and the majority of these patients die in the neonatal period. The only known treatment is liver transplant. The attached table discloses the correcting oligonucleotide base sequences for the UGT1 oligonucleotides of the invention.

Table 29
UGT1 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Crigler-Najjar syndrome 2 Leu15Arg CTG-CGG	GCAGGAGCAAAGGCGCCATGGCTGTGGAGTCCCAGGGCGG ACGCCCACTTGTCCTGGGCCTGCTGCTGTGTGTGCTGGGCC CAGTGGTGTCCCATGCTGGGAAGATACTGTTGATCCCAGT	3960
	ACTGGGATCAACAGTATCTTCCCAGCATGGGACACCACTGGG CCCAGCACACACAGCAGCAGGCCCAGGACAAGTGGGCGTCC GCCCTGGGACTCCACAGCCATGGCGCCTTTGCTCCTGC	3961
	CCTGGGCCTGCTGCTGT	3962
	ACAGCAGCAGGCCCAGG	3963
Crigler-Najjar syndrome 1 Gln49Term CAG-TAG	GGGAAGATACTGTTGATCCCAGTGGATGGCAGCCACTGGCT GAGCATGCTTGGGGCCATCCAGCAGCTGCAGCAGAGGGGAC ATGAAATAGTTGTCCTAGCACCTGACGCCTCGTTGTACA	3964
	TGTACAACGAGGCGTCAGGTGCTAGGACAACTATTTCATGTC CCCTCTGCTGCAGCTGCTGGATGGCCCCAAGCATGCTCAGC CAGTGGCTGCCATCCACTGGGATCAACAGTATCTTCCC	3965
	GGGCCATCCAGCAGCTG	3966
	CAGCTGCTGGATGGCCC	3967
Crigler-Najjar syndrome 1 Gly71Arg GGA-AGA	CAGCAGAGGGGACATGAAATAGTTGTCCTAGCACCTGACGCC TCGTTGTACATCAGAGACGGAGCATTTTACACCTTGAAGACGT ACCCTGTGCCATTCCAAAGGGAGGATGTGAAAGAGT	3968
	ACTCTTTCACATCCTCCCTTTGGAATGGCACAGGGTACGTCTT CAAGGTGTAAAATGCTCCGTCTCTGATGTACAACGAGGCGTC AGGTGCTAGGACAACTATTTCATGTCCCCTCTGCTG	3969
	TCAGAGACGGAGCATTT	3970
	AAATGCTCCGTCTCTGA	3971
Gilbert syndrome Pro229Gln CCG-CAG	GGGTGAAGAACATGCTCATTGCCTTTTCACAGAACTTTCTGTG CGACGTGGTTTATTCCCCGTATGCAACCCTTGCCTCAGAATT CCTTCAGAGAGAGGTGACTGTCCAGGACCTATTGAG	3972
	CTCAATAGGTCCTGGACAGTCACCTCTCTCTGAAGGAATTCT GAGGCAAGGGTTGCATACGGGGAATAAACCACGTCGCACAG AAAGTTCTGTGAAAAGGCAATGAGCATGTTCTTCACCC	3973
	TTATTCCCCGTATGCAA	3974
	TTGCATACGGGGAATAA	3975
Crigler-Najjar syndrome 1 Cys280Term TGC-TGA	TGTGAAGGATTACCCTAGGCCCATCATGCCCAATATGGTTTTT GTTGGTGGAATCAACTGCCTTCACCAAAATCCACTATCCCAG GTGTGTATTGGAGTGGGACTTTTACATGCGTATATT	3976
	AATACGCATGTAAAAGTCCCACTCCAATACACACCTGGGAT AGTGGATTTTGGTGAAGGCAGTTGATTCCACCAACAAAAACC ATATTGGGCATGATGGGCCTAGGGTAATCCTTCACA	3977

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATCAACTGCCTTCACCA	3978
	TGGTGAAGGCAGTTGAT	3979
Crigler-Najjar syndrome 1 Ala292Val GCC-GTC	ATCAAAGAATATGAGAAAAAATTAACTGAAAATTTTTCTTCTGG CTCTAGGAATTTGAAGCCTACATTAATGCTTCTGGAGAACATG GAATTGTGGTTTTCTCTTTGGGATCAATGGTCTC	3980
	GAGACCATTGATCCCAAAGAGAAAACCACAATTCCATGTTCTC CAGAAGCATTAATGTAGGCTTCAAATTCCTAGAGCCAGAAGAA AAATTTTCAGTTAATTTTTTCTCATATTCTTTGAT	3981
	ATTTGAAGCCTACATTA	3982
	TAATGTAGGCTTCAAAT	3983
Crigler-Najjar syndrome 1 Gly308Glu GGA-GAA	AGGAATTTGAAGCCTACATTAATGCTTCTGGAGAACATGGAAT TGTGGTTTTCTCTTTGGGATCAATGGTCTCAGAAATTCCAGAG AAGAAAGCTATGGCAATTGCTGATGCTTTGGGCAA	3984
	TTGCCCAAAGCATCAGCAATTGCCATAGCTTTCTTCTCTGGAA TTTCTGAGACCATTGATCCCAAAGAGAAAACCACAATTCCATG TTCTCCAGAAGCATTAATGTAGGCTTCAAATTCCT	3985
	CTCTTTGGGATCAATGG	3986
	CCATTGATCCCAAAGAG	3987
Crigler-Najjar syndrome 1 Gln331Term CAG-TAG	GTCTCAGAAATTCCAGAGAAGAAAGCTATGGCAATTGCTGAT GCTTTGGGCAAAATCCCTCAGACAGTAAGAAGATTCTATACCA TGGCCTCATATCTATTTTCACAGGAGCGCTAATCCC	3988
	GGGATTAGCGCTCCTGTGAAAATAGATATGAGGCCATGGTAT AGAATCTTCTTACTGTCTGAGGGATTTTGCCCAAAGCATCAGC AATTGCCATAGCTTTCTTCTCTGGAATTTCTGAGAC	3989
	AAATCCCTCAGACAGTA	3990
	TACTGTCTGAGGGATTT	3991
Crigler-Najjar syndrome 1 Trp335Term TGG-TGA	TCTAATCATATTATGTTCTTTCTTTACGTTCTGCTCTTTTTGCC CCTCCCAGGTCCTGTGGCGGTACACTGGAACCCGACCATCG AATCTTGCGAACAACACGATACTTGTTAAGTGGCTA	3992
	TAGCCACTTAACAAGTATCGTGTTGTTCGCAAGATTCGATGGT CGGGTTCCAGTGTACCGCCACAGGACCTGGGAGGGGCAAAA AGAGCAGAACGTAAAGAAAGAACATAATATGATTAGA	3993
	GTCCTGTGGCGGTACAC	3994
	GTGTACCGCCACAGGAC	3995
Crigler-Najjar syndrome 1 Gln357Arg CAA-CGA	ACACTGGAACCCGACCATCGAATCTTGCGAACAACACGATAC TTGTTAAGTGGCTACCCCAAAACGATCTGCTTGGTATGTTGG GCGGATTGGATGTATAGGTCAAACCAGGGTCAAATTA	3996
	TAATTTGACCCTGGTTTGACCTATACATCCAATCCGCCCAACA TACCAAGCAGATCGTTTTGGGGTAGCCACTTAACAAGTATCGT GTTGTTCGCAAGATTCGATGGTCGGGTTCCAGTGT	3997

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCTACCCCAAAACGATC	3998
	GATCGTTTTGGGGTAGC	3999
Crigler-Najjar syndrome 1 Gln357Term CAA-TAA	TACACTGGAACCCGACCATCGAATCTTGCGAACAACACGATA CTTGTTAAGTGGCTACCCCAAAACGATCTGCTTGGTATGTTG GGCGGATTGGATGTATAGGTCAAACCAGGGTCAAATT	4000
	AATTTGACCCTGGTTTGACCTATACATCCAATCCGCCCAACAT ACCAAGCAGATCGTTTTGGGGTAGCCACTTAACAAGTATCGT GTTGTTCGCAAGATTCGATGGTCGGGTTCCAGTGTA	4001
	GGCTACCCCAAAACGAT	4002
	ATCGTTTTGGGGTAGCC	4003
Gilbert syndrome Arg367Gly CGT-GGT	AACTCAGAGATGTAACTGCTGACATCCTCCCTATTTTGCATCT CAGGTCACCCGATGACCCGTGCCTTTATCACCCATGCTGGTT CCCATGGTGTTTATGAAAGCATATGCAATGGCGTTC	4004
	GAACGCCATTGCATATGCTTTCATAAACACCATGGGAACCAG CATGGGTGATAAAGGCACGGGTCATCGGGTGACCTGAGATG CAAAATAGGGAGGATGTCAGCAGTTACATCTCTGAGTT	4005
	CGATGACCCGTGCCTTT	4006
	AAAGGCACGGGTCATCG	4007
Crigler-Najjar syndrome 1 Ala368Thr GCC-ACC	TCAGAGATGTAACTGCTGACATCCTCCCTATTTTGCATCTCAG GTCACCCGATGACCCGTGCCTTTATCACCCATGCTGGTTCCC ATGGTGTTTATGAAAGCATATGCAATGGCGTTCCCA	4008
	TGGGAACGCCATTGCATATGCTTTCATAAACACCATGGGAAC CAGCATGGGTGATAAAGGCACGGGTCATCGGGTGACCTGAG ATGCAAAATAGGGAGGATGTCAGCAGTTACATCTCTGA	4009
	TGACCCGTGCCTTTATC	4010
	GATAAAGGCACGGGTCA	4011
Crigler-Najjar syndrome 1 Ser375Phe TCC-TTC	CCTCCCTATTTTGCATCTCAGGTCACCCGATGACCCGTGCCT TTATCACCCATGCTGGTTCCCATGGTGTTTATGAAAGCATATG CAATGGCGTTCCCATGGTGATGATGCCCTTGTTTGG	4012
	CCAAACAAGGGCATCATCACCATGGGAACGCCATTGCATATG CTTTCATAAACACCATGGGAACCAGCATGGGTGATAAAGGCA CGGGTCATCGGGTGACCTGAGATGCAAAATAGGGAGG	4013
	TGCTGGTTCCCATGGTG	4014
	CACCATGGGAACCAGCA	4015
Crigler-Najjar syndrome 1 Ser381Arg AGC-AGG	AGGTCACCCGATGACCCGTGCCTTTATCACCCATGCTGGTTC CCATGGTGTTTATGAAAGCATATGCAATGGCGTTCCCATGGT GATGATGCCCTTGTTTGGTGATCAGATGGACAATGCA	4016
	TGCATTGTCCATCTGATCACCAAACAAGGGCATCATCCCCAT GGGAACGCCATTGCATATGCTTTCATAAACACCATGGGAACC AGCATGGGTGATAAAGGCACGGGTCATCGGGTGACCT	4017

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TATGAAAGCATATGCAA	4018
	TTGCATATGCTTTCATA	4019
Crigler-Najjar syndrome 1 Ala401Pro GCA-CCA	AGCATATGCAATGGCGTTCCCATGGTGATGATGCCCTTGTTT GGTGATCAGATGGACAATGCAAAGCGCATGGAGACTAAGGG AGCTGGAGTGACCCTGAATGTTCTGGAAATGACTTCTG	4020
	CAGAAGTCATTTCCAGAACATTCAGGGTCACTCCAGCTCCCT TAGTCTCCATGCGCTTTGCATTGTCCATCTGATCACCAAACAA GGGCATCATCACCATGGGAACGCCATTGCATATGCT	4021
	TGGACAATGCAAAGCGC	4022
	GCGCTTTGCATTGTCCA	4023
Crigler-Najjar syndrome 1 Lys428Glu AAA-GAA	GGAGCTGGAGTGACCCTGAATGTTCTGGAAATGACTTCTGAA GATTTAGAAAATGCTCTAAAAGCAGTCATCAATGACAAAAGGT AAGAAAGAAGATACAGAAGAATACTTTGGTCATGGC	4024
	GCCATGACCAAAGTATTCTTCTGTATCTTCTTTCTTACCTTTTG TCATTGATGACTGCTTTTAGAGCATTTTCTAAATCTTCAGAAGT CATTTCCAGAACATTCAGGGTCACTCCAGCTCC	4025
	ATGCTCTAAAAGCAGTC	4026
	GACTGCTTTTAGAGCAT	4027
Crigler-Najjar syndrome 1 Tyr486Asp TAC-GAC	ATGAGGCACAAGGGCGCGCCACACCTGCGCCCCGCAGCCC ACGACCTCACCTGGTACCAGTACCATTCCTTGGACGTGATTG GTTTCCTCTTGGCCGTCGTGCTGACAGTGGCCTTCATCA	4028
	TGATGAAGGCCACTGTCAGCACGACGGCCAAGAGGAAACCA ATCACGTCCAAGGAATGGTACTGGTACCAGGTGAGGTCGTG GGCTGCGGGGCGCAGGTGTGGCGCGCCCTTGTGCCTCAT	4029
	GGTACCAGTACCATTCC	4030
	GGAATGGTACTGGTACC	4031
Crigler-Najjar syndrome 1 Ser488Phe TCC-TTC	ACAAGGGCGCGCCACACCTGCGCCCCGCAGCCCACGACCT CACCTGGTACCAGTACCATTCCTTGGACGTGATTGGTTTCCT CTTGGCCGTCGTGCTGACAGTGGCCTTCATCACCTTTAA	4032
	TTAAAGGTGATGAAGGCCACTGTCAGCACGACGGCCAAGAG GAAACCAATCACGTCCAAGGAATGGTACTGGTACCAGGTGAG GTCGTGGGCTGCGGGGCGCAGGTGTGGCGCGCCCTTGT	4033
	GTACCATTCCTTGGACG	4034
	CGTCCAAGGAATGGTAC	4035

EXAMPLE 23
Alzheimer's Disease - Amyloid precursor protein (APP)

Over the past few decades Alzheimer's disease (AD), once considered a rare disorder, has become recognized as a major public health problem. Although there is no agreement on the exact prevalence of Alzheimer's disease, in part due to difficulties of diagnosis, studies consistently point to an exponential rise in prevalence of this disease with age. After age 65, the percentage of affected people approximately doubles with every decade of life, regardless of definition. Among people age 85 or older, studies suggest that 25 to 35 percent have dementia, including Alzheimer's disease; one study reports that 47.2 percent of people over age 85 have Alzheimer's disease, exclusive of other dementias.

Alzheimer's disease progressively destroys memory, reason, judgment, language, and, eventually, the ability to carry out even the simplest tasks. Anatomic changes associated with Alzheimer's disease begin in the entorhinal cortex, proceed to the hippocampus, and then gradually spread to other regions, particularly the cerebral cortex. Chief among such anatomic changes are the presence of characteristic extracellular plaques and internal neurofibrillary tangles.

At least four genes have been identified to date that contribute to development of Alzheimer's disease: AD1 is caused by mutations in the amyloid precursor gene (APP); AD2 is associated with a particular allele of APOE (see Example 20); AD3 is caused by mutation in a gene encoding a 7-transmembrane domain protein, presenilin-1 (PSEN1), and AD4 is caused by mutation in a gene that encodes a similar 7-transmembrane domain protein, presenilin-2 (PSEN2). The attached table discloses the correcting oligonucleotide base sequences for the APP oligonucleotides of the invention.

Table 30
APP Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Glu665Asp GAG-GAC	CTGCATACTTTAATTATGATGTAATACAGGTTCTGGGTTGACA AATATCAAGACGGAGGA**G**ATCTCTGAAGTGAAGATGGATGCA GAATTCCGACATGACTCAGGATATGAAGTTCATCAT	4036
	ATGATGAACTTCATATCCTGAGTCATGTCGGAATTCTGCATCC ATCTTCACTTCAGAGAT**C**TCCTCCGTCTTGATATTTGTCAACC CAGAACCTGTATTACATCATAATTAAAGTATGCAG	4037
	ACGGAGGA**G**ATCTCTGA	4038
	TCAGAGAT**C**TCCTCCGT	4039
Alzheimer disease Ala692Gly GCA-GGA	ATTATATTGCATTTAGAAATTAAAATTCTTTTTCTTAATTTGTTTT CAAGGTGTTCTTTG**C**AGAAGATGTGGGTTCAAACAAAGGTGC AATCATTGGACTCATGGTGGGCGGTGTTGTCAT	4040

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATGACAACACCGCCCACCATGAGTCCAATGATTGCACCTTTG TTTGAACCCACATCTTCTGCAAAGAACACCTTGAAAACAAATT AAGAAAAAGAATTTTAATTTCTAAATGCAATATAAT	4041
	GTTCTTTGCAGAAGATG	4042
	CATCTTCTGCAAAGAAC	4043
Alzheimer disease Glu693Gln GAA-CAA	TATATTGCATTTAGAAATTAAAATTCTTTTTCTTAATTTGTTTTC AAGGTGTTCTTTGCAGAAGATGTGGGTTCAAACAAAGGTGCA ATCATTGGACTCATGGTGGGCGGTGTTGTCATAG	4044
	CTATGACAACACCGCCCACCATGAGTCCAATGATTGCACCTT TGTTTGAACCCACATCTTCTGCAAAGAACACCTTGAAAACAAA TTAAGAAAAAGAATTTTAATTTCTAAATGCAATATA	4045
	TCTTTGCAGAAGATGTG	4046
	CACATCTTCTGCAAAGA	4047
Alzheimer disease Glu693Gly GAA-GGA	ATATTGCATTTAGAAATTAAAATTCTTTTTCTTAATTTGTTTTCA AGGTGTTCTTTGCAGAAGATGTGGGTTCAAACAAAGGTGCAA TCATTGGACTCATGGTGGGCGGTGTTGTCATAGC	4048
	GCTATGACAACACCGCCCACCATGAGTCCAATGATTGCACCT TTGTTTGAACCCACATCTTCTGCAAAGAACACCTTGAAAACAA ATTAAGAAAAAGAATTTTAATTTCTAAATGCAATAT	4049
	CTTTGCAGAAGATGTGG	4050
	CCACATCTTCTGCAAAG	4051
Alzheimer disease Ala713Thr GCG-ACG	GAAGATGTGGGTTCAAACAAAGGTGCAATCATTGGACTCATG GTGGGCGGTGTTGTCATAGCGACAGTGATCGTCATCACCTTG GTGATGCTGAAGAAGAAACAGTACACATCCATTCATC	4052
	GATGAATGGATGTGTACTGTTTCTTCTTCAGCATCACCAAGGT GATGACGATCACTGTCGCTATGACAACACCGCCCACCATGAG TCCAATGATTGCACCTTTGTTTGAACCCACATCTTC	4053
	TTGTCATAGCGACAGTG	4054
	CACTGTCGCTATGACAA	4055
Schizophrenia Ala713Val GCG-GTG	AAGATGTGGGTTCAAACAAAGGTGCAATCATTGGACTCATGG TGGGCGGTGTTGTCATAGCGACAGTGATCGTCATCACCTTGG TGATGCTGAAGAAGAAACAGTACACATCCATTCATCA	4056
	TGATGAATGGATGTGTACTGTTTCTTCTTCAGCATCACCAAGG TGATGACGATCACTGTCGCTATGACAACACCGCCCACCATGA GTCCAATGATTGCACCTTTGTTTGAACCCACATCTT	4057
	TGTCATAGCGACAGTGA	4058
	TCACTGTCGCTATGACA	4059
Alzheimer disease Val715Met GTG-ATG	GTGGGTTCAAACAAAGGTGCAATCATTGGACTCATGGTGGGC GGTGTTGTCATAGCGACAGTGATCGTCATCACCTTGGTGATG CTGAAGAAGAAACAGTACACATCCATTCATCATGGTG	4060
	CACCATGATGAATGGATGTGTACTGTTTCTTCTTCAGCATCAC CAAGGTGATGACGATCACTGTCGCTATGACAACACCGCCCAC CATGAGTCCAATGATTGCACCTTTGTTTGAACCCAC	4061
	TAGCGACAGTGATCGTC	4062

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GACGATCACTGTCGCTA	4063
Alzheimer disease Ile716Val ATC-GTC	GGTTCAAACAAAGGTGCAATCATTGGACTCATGGTGGGCGGT GTTGTCATAGCGACAGTGATCGTCATCACCTTGGTGATGCTG AAGAAGAAACAGTACACATCCATTCATCATGGTGTGG	4064
	CCACACCATGATGAATGGATGTGTACTGTTTCTTCTTCAGCAT CACCAAGGTGATGACGATCACTGTCGCTATGACAACACCGCC CACCATGAGTCCAATGATTGCACCTTTGTTTGAACC	4065
	CGACAGTGATCGTCATC	4066
	GATGACGATCACTGTCG	4067
Alzheimer disease Val717Gly GTC-GGC	CAAACAAAGGTGCAATCATTGGACTCATGGTGGGCGGTGTTG TCATAGCGACAGTGATCGTCATCACCTTGGTGATGCTGAAGA AGAAACAGTACACATCCATTCATCATGGTGTGGTGGA	4068
	TCCACCACACCATGATGAATGGATGTGTACTGTTTCTTCTTCA GCATCACCAAGGTGATGACGATCACTGTCGCTATGACAACAC CGCCCACCATGAGTCCAATGATTGCACCTTTGTTTG	4069
	AGTGATCGTCATCACCT	4070
	AGGTGATGACGATCACT	4071
Alzheimer disease Val717Ile GTC-ATC	TCAAACAAAGGTGCAATCATTGGACTCATGGTGGGCGGTGTT GTCATAGCGACAGTGATCGTCATCACCTTGGTGATGCTGAAG AAGAAACAGTACACATCCATTCATCATGGTGTGGTGG	4072
	CCACCACACCATGATGAATGGATGTGTACTGTTTCTTCTTCAG CATCACCAAGGTGATGACGATCACTGTCGCTATGACAACACC GCCCACCATGAGTCCAATGATTGCACCTTTGTTTGA	4073
	CAGTGATCGTCATCACC	4074
	GGTGATGACGATCACTG	4075
Alzheimer disease Val717Phe GTC-TTC	TCAAACAAAGGTGCAATCATTGGACTCATGGTGGGCGGTGTT GTCATAGCGACAGTGATCGTCATCACCTTGGTGATGCTGAAG AAGAAACAGTACACATCCATTCATCATGGTGTGGTGG	4076
	CCACCACACCATGATGAATGGATGTGTACTGTTTCTTCTTCAG CATCACCAAGGTGATGACGATCACTGTCGCTATGACAACACC GCCCACCATGAGTCCAATGATTGCACCTTTGTTTGA	4077
	CAGTGATCGTCATCACC	4078
	GGTGATGACGATCACTG	4079
Alzheimer disease Leu723Pro CTG-CCG	TTGGACTCATGGTGGGCGGTGTTGTCATAGCGACAGTGATCG TCATCACCTTGGTGATGCTGAAGAAGAAACAGTACACATCCAT TCATCATGGTGTGGTGGAGGTAGGTAAACTTGACTG	4080
	CAGTCAAGTTTACCTACCTCCACCACACCATGATGAATGGAT GTGTACTGTTTCTTCTTCAGCATCACCAAGGTGATGACGATCA CTGTCGCTATGACAACACCGCCCACCATGAGTCCAA	4081
	GGTGATGCTGAAGAAGA	4082
	TCTTCTTCAGCATCACC	4083

EXAMPLE 24
Alzheimer's Disease - presenilin-1 (PSEN1)

The attached table discloses the correcting oligonucleotide base sequences for the PSEN1 oligonucleotides of the invention.

Table 31
PSEN1 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Ala79Val GCC-GTC	CCCGGCAGGTGGTGGAGCAAGATGAGGAAGAAGATGAGGAG CTGACATTGAAATATGGCG**C**CAAGCATGTGATCATGCTCTTTG TCCCTGTGACTCTCTGCATGGTGGTGGTCGTGGCTAC	4084
	GTAGCCACGACCACCACCATGCAGAGAGTCACAGGGACAAA GAGCATGATCACATGCTTG**G**CGCCATATTTCAATGTCAGCTC CTCATCTTCTTCCTCATCTTGCTCCACCACCTGCCGGG	4085
	ATATGGCG**C**CAAGCATG	4086
	CATGCTTG**G**CGCCATAT	4087
Alzheimer disease Val82Leu tGTG-CTG	GTGGTGGAGCAAGATGAGGAAGAAGATGAGGAGCTGACATT GAAATATGGCGCCAAGCAT**G**TGATCATGCTCTTTGTCCCTGT GACTCTCTGCATGGTGGTGGTCGTGGCTACCATTAAGT	4088
	ACTTAATGGTAGCCACGACCACCACCATGCAGAGAGTCACAG GGACAAAGAGCATGATCA**C**ATGCTTGGCGCCATATTTCAATG TCAGCTCCTCATCTTCTTCCTCATCTTGCTCCACCAC	4089
	CCAAGCAT**G**TGATCATG	4090
	CATGATCA**C**ATGCTTGG	4091
Alzheimer disease Val96Phe gGTC-TTC	AAATATGGCGCCAAGCATGTGATCATGCTCTTTGTCCCTGTG ACTCTCTGCATGGTGGTG**G**TCGTGGCTACCATTAAGTCAGTC AGCTTTTATACCCGGAAGGATGGGCAGCTGTACGTAT	4092
	ATACGTACAGCTGCCCATCCTTCCGGGTATAAAAGCTGACTG ACTTAATGGTAGCCACGA**C**CACCACCATGCAGAGAGTCACAG GGACAAAGAGCATGATCACATGCTTGGCGCCATATTT	4093
	TGGTGGTG**G**TCGTGGCT	4094
	AGCCACGA**C**CACCACCA	4095
Alzheimer disease Phe105Leu TTTt-TTG	CTTTGTCCCTGTGACTCTCTGCATGGTGGTGGTCGTGGCTAC CATTAAGTCAGTCAGCTTT**T**TATACCCGGAAGGATGGGCAGCT GTACGTATGAGTTTTGTTTTATTATTCTCAAAGCCAG	4096
	CTGGCTTTGAGAATAATAAAACAAAACTCATACGTACAGCTGC CCATCCTTCCGGGTATA**A**AAGCTGACTGACTTAATGGTAGCC ACGACCACCACCATGCAGAGAGTCACAGGGACAAAG	4097
	GTCAGCTTT**T**TATACCCG	4098
	CGGGTATA**A**AAGCTGAC	4099

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Thr116Asn ACC-AAC	TGGTGATCTCCATTAACACTGACCTAGGGCTTTTGTGTTTGTT TTATTGTAGAATCTATA**C**CCCATTCACAGAAGATACCGAGACT GTGGGCCAGAGAGCCCTGCACTCAATTCTGAATGC	4100
	GCATTCAGAATTGAGTGCAGGGCTCTCTGGCCCACAGTCTCG GTATCTTCTGTGAATGGG**G**TATAGATTCTACAATAAAACAAAC ACAAAAGCCCTAGGTCAGTGTTAATGGAGATCACCA	4101
	AATCTATA**C**CCCATTCA	4102
	TGAATGGG**G**TATAGATT	4103
Alzheimer disease Pro117Leu CCA-CTA	TGATCTCCATTAACACTGACCTAGGGCTTTTGTGTTTGTTTTAT TGTAGAATCTATACCC**C**ATTCACAGAAGATACCGAGACTGTG GGCCAGAGAGCCCTGCACTCAATTCTGAATGCTGC	4104
	GCAGCATTCAGAATTGAGTGCAGGGCTCTCTGGCCCACAGTC TCGGTATCTTCTGTGAAT**G**GGGTATAGATTCTACAATAAAACA AACACAAAAGCCCTAGGTCAGTGTTAATGGAGATCA	4105
	CTATACCC**C**ATTCACAG	4106
	CTGTGAAT**G**GGGTATAG	4107
Alzheimer disease Glu120Asp GAAg-GAT	TAACACTGACCTAGGGCTTTTGTGTTTGTTTTATTGTAGAATCT ATACCCCATTCACAGA**A**GATACCGAGACTGTGGGCCAGAGAG CCCTGCACTCAATTCTGAATGCTGCCATCATGATC	4108
	GATCATGATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCTG GCCCACAGTCTCGGTATC**T**TCTGTGAATGGGGTATAGATTCT ACAATAAAACAAACACAAAAGCCCTAGGTCAGTGTTA	4109
	TTCACAGA**A**GATACCGA	4110
	TCGGTATC**T**TCTGTGAA	4111
Alzheimer disease Glu120Asp GAAg-GAC	TAACACTGACCTAGGGCTTTTGTGTTTGTTTTATTGTAGAATCT ATACCCCATTCACAGA**A**GATACCGAGACTGTGGGCCAGAGAG CCCTGCACTCAATTCTGAATGCTGCCATCATGATC	4112
	GATCATGATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCTG GCCCACAGTCTCGGTATC**T**TCTGTGAATGGGGTATAGATTCT ACAATAAAACAAACACAAAAGCCCTAGGTCAGTGTTA	4113
	TTCACAGA**A**GATACCGA	4114
	TCGGTATC**T**TCTGTGAA	4115
Alzheimer disease Glu120Lys aGAA-AAA	ATTAACACTGACCTAGGGCTTTTGTGTTTGTTTTATTGTAGAAT CTATACCCCATTCACA**G**AAGATACCGAGACTGTGGGCCAGAG AGCCCTGCACTCAATTCTGAATGCTGCCATCATGA	4116
	TCATGATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCTGGC CCACAGTCTCGGTATCTT**C**TGTGAATGGGGTATAGATTCTACA ATAAAACAAACACAAAAGCCCTAGGTCAGTGTTAAT	4117
	CATTCACA**G**AAGATACC	4118
	GGTATCTT**C**TGTGAATG	4119
Alzheimer disease Glu123Lys cGAG-AAG	GACCTAGGGCTTTTGTGTTTGTTTTATTGTAGAATCTATACCC CATTCACAGAAGATACC**G**AGACTGTGGGCCAGAGAGCCCTG CACTCAATTCTGAATGCTGCCATCATGATCAGTGTCA	4120

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGACACTGATCATGATGGCAGCATTCAGAATTGAGTGCAGGG CTCTCTGGCCCACAGTCTCGGTATCTTCTGTGAATGGGGTAT AGATTCTACAATAAAACAAACACAAAAGCCCTAGGTC	4121
	AAGATACCGAGACTGTG	4122
	CACAGTCTCGGTATCTT	4123
Alzheimer disease Asn135Asp gAAT-GAT	TATACCCCATTCACAGAAGATACCGAGACTGTGGGCCAGAGA GCCCTGCACTCAATTCTGAATGCTGCCATCATGATCAGTGTC ATTGTTGTCATGACTATCCTCCTGGTGGTTCTGTATA	4124
	TATACAGAACCACCAGGAGGATAGTCATGACAACAATGACAC TGATCATGATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCT GGCCCACAGTCTCGGTATCTTCTGTGAATGGGGTATA	4125
	CAATTCTGAATGCTGCC	4126
	GGCAGCATTCAGAATTG	4127
Alzheimer disease Met139Ile ATGa-ATA	AGAAGATACCGAGACTGTGGGCCAGAGAGCCCTGCACTCAA TTCTGAATGCTGCCATCATGATCAGTGTCATTGTTGTCATGAC TATCCTCCTGGTGGTTCTGTATAAATACAGGTGCTAT	4128
	ATAGCACCTGTATTTATACAGAACCACCAGGAGGATAGTCATG ACAACAATGACACTGATCATGATGGCAGCATTCAGAATTGAGT GCAGGGCTCTCTGGCCCACAGTCTCGGTATCTTCT	4129
	GCCATCATGATCAGTGT	4130
	ACACTGATCATGATGGC	4131
Alzheimer disease Met139Lys ATG-AAG	CAGAAGATACCGAGACTGTGGGCCAGAGAGCCCTGCACTCA ATTCTGAATGCTGCCATCATGATCAGTGTCATTGTTGTCATGA CTATCCTCCTGGTGGTTCTGTATAAATACAGGTGCTA	4132
	TAGCACCTGTATTTATACAGAACCACCAGGAGGATAGTCATGA CAACAATGACACTGATCATGATGGCAGCATTCAGAATTGAGT GCAGGGCTCTCTGGCCCACAGTCTCGGTATCTTCTG	4133
	TGCCATCATGATCAGTG	4134
	CACTGATCATGATGGCA	4135
Alzheimer disease Met139Thr ATG-ACG	CAGAAGATACCGAGACTGTGGGCCAGAGAGCCCTGCACTCA ATTCTGAATGCTGCCATCATGATCAGTGTCATTGTTGTCATGA CTATCCTCCTGGTGGTTCTGTATAAATACAGGTGCTA	4136
	TAGCACCTGTATTTATACAGAACCACCAGGAGGATAGTCATGA CAACAATGACACTGATCATGATGGCAGCATTCAGAATTGAGT GCAGGGCTCTCTGGCCCACAGTCTCGGTATCTTCTG	4137
	TGCCATCATGATCAGTG	4138
	CACTGATCATGATGGCA	4139
Alzheimer disease Met139Val cATG-GTG	ACAGAAGATACCGAGACTGTGGGCCAGAGAGCCCTGCACTC AATTCTGAATGCTGCCATCATGATCAGTGTCATTGTTGTCATG ACTATCCTCCTGGTGGTTCTGTATAAATACAGGTGCT	4140
	AGCACCTGTATTTATACAGAACCACCAGGAGGATAGTCATGA CAACAATGACACTGATCATGATGGCAGCATTCAGAATTGAGT GCAGGGCTCTCTGGCCCACAGTCTCGGTATCTTCTGT	4141
	CTGCCATCATGATCAGT	4142

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACTGATCATGATGGCAG	4143
Alzheimer disease Ile143Phe cATT-TTT	GAGACTGTGGGCCAGAGAGCCCTGCACTCAATTCTGAATGCT GCCATCATGATCAGTGTCATTGTTGTCATGACTATCCTCCTGG TGGTTCTGTATAAATACAGGTGCTATAAGGTGAGCA	4144
	TGCTCACCTTATAGCACCTGTATTTATACAGAACCACCAGGAG GATAGTCATGACAACAATGACACTGATCATGATGGCAGCATTC AGAATTGAGTGCAGGGCTCTCTGGCCCACAGTCTC	4145
	TCAGTGTCATTGTTGTC	4146
	GACAACAATGACACTGA	4147
Alzheimer disease Ile143Thr ATT-ACT	AGACTGTGGGCCAGAGAGCCCTGCACTCAATTCTGAATGCTG CCATCATGATCAGTGTCATTGTTGTCATGACTATCCTCCTGGT GGTTCTGTATAAATACAGGTGCTATAAGGTGAGCAT	4148
	ATGCTCACCTTATAGCACCTGTATTTATACAGAACCACCAGGA GGATAGTCATGACAACAATGACACTGATCATGATGGCAGCAT TCAGAATTGAGTGCAGGGCTCTCTGGCCCACAGTCT	4149
	CAGTGTCATTGTTGTCA	4150
	TGACAACAATGACACTG	4151
Alzheimer disease Met146Ile ATGa-ATA	CCAGAGAGCCCTGCACTCAATTCTGAATGCTGCCATCATGAT CAGTGTCATTGTTGTCATGACTATCCTCCTGGTGGTTCTGTAT AAATACAGGTGCTATAAGGTGAGCATGAGACACAGA	4152
	TCTGTGTCTCATGCTCACCTTATAGCACCTGTATTTATACAGA ACCACCAGGAGGATAGTCATGACAACAATGACACTGATCATG ATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCTGG	4153
	GTTGTCATGACTATCCT	4154
	AGGATAGTCATGACAAC	4155
Alzheimer disease Met146Ile ATGa-ATC	CCAGAGAGCCCTGCACTCAATTCTGAATGCTGCCATCATGAT CAGTGTCATTGTTGTCATGACTATCCTCCTGGTGGTTCTGTAT AAATACAGGTGCTATAAGGTGAGCATGAGACACAGA	4156
	TCTGTGTCTCATGCTCACCTTATAGCACCTGTATTTATACAGA ACCACCAGGAGGATAGTCATGACAACAATGACACTGATCATG ATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCTGG	4157
	GTTGTCATGACTATCCT	4158
	AGGATAGTCATGACAAC	4159
Alzheimer disease Met146Leu cATG-TTG	GGCCAGAGAGCCCTGCACTCAATTCTGAATGCTGCCATCATG ATCAGTGTCATTGTTGTCATGACTATCCTCCTGGTGGTTCTGT ATAAATACAGGTGCTATAAGGTGAGCATGAGACACA	4160
	TGTGTCTCATGCTCACCTTATAGCACCTGTATTTATACAGAAC CACCAGGAGGATAGTCATGACAACAATGACACTGATCATGAT GGCAGCATTCAGAATTGAGTGCAGGGCTCTCTGGCC	4161
	TTGTTGTCATGACTATC	4162
	GATAGTCATGACAACAA	4163
Alzheimer disease Met146Val cATG-GTG	GGCCAGAGAGCCCTGCACTCAATTCTGAATGCTGCCATCATG ATCAGTGTCATTGTTGTCATGACTATCCTCCTGGTGGTTCTGT ATAAATACAGGTGCTATAAGGTGAGCATGAGACACA	4164

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGTGTCTCATGCTCACCTTATAGCACCTGTATTTATACAGAAC CACCAGGAGGATAGTCATGACAACAATGACACTGATCATGAT GGCAGCATTCAGAATTGAGTGCAGGGCTCTCTGGCC	4165
	TTGTTGTCATGACTATC	4166
	GATAGTCATGACAACAA	4167
Alzheimer disease Thr147Ile ACT-ATT	AGAGAGCCCTGCACTCAATTCTGAATGCTGCCATCATGATCA GTGTCATTGTTGTCATGACTATCCTCCTGGTGGTTCTGTATAA ATACAGGTGCTATAAGGTGAGCATGAGACACAGATC	4168
	GATCTGTGTCTCATGCTCACCTTATAGCACCTGTATTTATACA GAACCACCAGGAGGATAGTCATGACAACAATGACACTGATCA TGATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCT	4169
	TGTCATGACTATCCTCC	4170
	GGAGGATAGTCATGACA	4171
Alzheimer disease His163Arg CAT-CGT	CTTTTTAAGGGTTGTGGGACCTGTTAATTATATTGAAATGCTTT CTTTTCTAGGTCATCCATGCCTGGCTTATTATATCATCTCTATT GTTGCTGTTCTTTTTTTCATTCATTTACTTGGG	4172
	CCCAAGTAAATGAATGAAAAAAAGAACAGCAACAATAGAGATG ATATAATAAGCCAGGCATGGATGACCTAGAAAAGAAAGCATTT CAATATAATTAACAGGTCCCACAACCCTTAAAAAG	4173
	GGTCATCCATGCCTGGC	4174
	GCCAGGCATGGATGACC	4175
Alzheimer disease His163Tyr cCAT-TAT	ACTTTTTAAGGGTTGTGGGACCTGTTAATTATATTGAAATGCTT TCTTTTCTAGGTCATCCATGCCTGGCTTATTATATCATCTCTAT TGTTGCTGTTCTTTTTTTCATTCATTTACTTGG	4176
	CCAAGTAAATGAATGAAAAAAAGAACAGCAACAATAGAGATGA TATAATAAGCCAGGCATGGATGACCTAGAAAAGAAAGCATTTC AATATAATTAACAGGTCCCACAACCCTTAAAAAGT	4177
	AGGTCATCCATGCCTGG	4178
	CCAGGCATGGATGACCT	4179
Alzheimer disease Trp165Cys TGGc-TGC	AGGGTTGTGGGACCTGTTAATTATATTGAAATGCTTTCTTTTCT AGGTCATCCATGCCTGGCTTATTATATCATCTCTATTGTTGCT GTTCTTTTTTTCATTCATTTACTTGGGGTAAGTT	4180
	AACTTACCCCAAGTAAATGAATGAAAAAAAGAACAGCAACAAT AGAGATGATATAATAAGCCAGGCATGGATGACCTAGAAAAGA AAGCATTTCAATATAATTAACAGGTCCCACAACCCT	4181
	CATGCCTGGCTTATTAT	4182
	ATAATAAGCCAGGCATG	4183
Alzheimer disease Ser169Leu TCA-TTA	ACCTGTTAATTATATTGAAATGCTTTCTTTTCTAGGTCATCCAT GCCTGGCTTATTATATCATCTCTATTGTTGCTGTTCTTTTTTTC ATTCATTTACTTGGGGTAAGTTGTGAAATTTTT	4184
	AAAAATTTCACAACTTACCCCAAGTAAATGAATGAAAAAAAGAA CAGCAACAATAGAGATGATAAATAAGCCAGGCATGGATGAC CTAGAAAAGAAAGCATTTCAATATAATTAACAGGT	4185
	TATTATATCATCTCTAT	4186

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATAGAGATGATATAATA	4187
Alzheimer disease Leu171Pro CTA-CCA	TAATTATATTGAAATGCTTTCTTTTCTAGGTCATCCATGCCTGG CTTATTATATCATCTCTATTGTTGCTGTTCTTTTTTTCATTCATT TACTTGGGGTAAGTTGTGAAATTTTTGGTCTG	4188
	CAGACCAAAAATTTCACAACTTACCCCAAGTAAATGAATGAAA AAAAGAACAGCAACAATAGAGATGATATAATAAGCCAGGCAT GGATGACCTAGAAAAGAAAGCATTTCAATATAATTA	4189
	ATCATCTCTATTGTTGC	4190
	GCAACAATAGAGATGAT	4191
Alzheimer disease Leu173Trp TTG-TGG	TATTGAAATGCTTTCTTTTCTAGGTCATCCATGCCTGGCTTATT ATATCATCTCTATTGTTGCTGTTCTTTTTTTCATTCATTTACTTG GGGTAAGTTGTGAAATTTTTGGTCTGTCTTTC	4192
	GAAAGACAGACCAAAAATTTCACAACTTACCCCAAGTAAATGA ATGAAAAAAAGAACAGCAACAATAGAGATGATATAATAAGCCA GGCATGGATGACCTAGAAAAGAAAGCATTTCAATA	4193
	TCTATTGTTGCTGTTCT	4194
	AGAACAGCAACAATAGA	4195
Alzheimer disease Gly209Arg gGGA-AGA	TATAACGTTGCTGTGGACTACATTACTGTTGCACTCCTGATCT GGAATTTTGGTGTGGTGGGAATGATTTCCATTCACTGGAAAG GTCCACTTCGACTCCAGCAGGCATATCTCATTATGA	4196
	TCATAATGAGATATGCCTGCTGGAGTCGAAGTGGACCTTTCC AGTGAATGGAAATCATTCCCACCACACCAAAATTCCAGATCAG GAGTGCAACAGTAATGTAGTCCACAGCAACGTTATA	4197
	GTGTGGTGGGAATGATT	4198
	AATCATTCCCACCACAC	4199
Alzheimer disease Gly209Val GGA-GTA	ATAACGTTGCTGTGGACTACATTACTGTTGCACTCCTGATCTG GAATTTTGGTGTGGTGGGAATGATTTCCATTCACTGGAAAGGT CCACTTCGACTCCAGCAGGCATATCTCATTATGAT	4200
	ATCATAATGAGATATGCCTGCTGGAGTCGAAGTGGACCTTTC CAGTGAATGGAAATCATTCCCACCACACCAAAATTCCAGATCA GGAGTGCAACAGTAATGTAGTCCACAGCAACGTTAT	4201
	TGTGGTGGGAATGATTT	4202
	AAATCATTCCCACCACA	4203
Alzheimer disease Ile213Thr ATT-ACT	TGGACTACATTACTGTTGCACTCCTGATCTGGAATTTTGGTGT GGTGGGAATGATTTCCATTCACTGGAAAGGTCCACTTCGACT CCAGCAGGCATATCTCATTATGATTAGTGCCCTCAT	4204
	ATGAGGGCACTAATCATAATGAGATATGCCTGCTGGAGTCGA AGTGGACCTTTCCAGTGAATGGAAATCATTCCCACCACACCA AAATTCCAGATCAGGAGTGCAACAGTAATGTAGTCCA	4205
	GATTTCCATTCACTGGA	4206
	TCCAGTGAATGGAAATC	4207
Alzheimer disease Leu219Pro CTT-CCT	CACTCCTGATCTGGAATTTTGGTGTGGTGGGAATGATTTCCAT TCACTGGAAAGGTCCACTTCGACTCCAGCAGGCATATCTCAT TATGATTAGTGCCCTCATGGCCCTGGTGTTTATCAA	4208

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTGATAAACACCAGGGCCATGAGGGCACTAATCATAATGAGA TATGCCTGCTGGAGTCGAAGTGGACCTTTCCAGTGAATGGAA ATCATTCCCACCACACCAAAATTCCAGATCAGGAGTG	4209
	AGGTCCACTTCGACTCC	4210
	GGAGTCGAAGTGGACCT	4211
Alzheimer disease Ala231Thr tGCC-ACC	ATTTCCATTCACTGGAAAGGTCCACTTCGACTCCAGCAGGCA TATCTCATTATGATTAGTGCCCTCATGGCCCTGGTGTTTATCA AGTACCTCCCTGAATGGACTGCGTGGCTCATCTTGG	4212
	CCAAGATGAGCCACGCAGTCCATTCAGGGAGGTACTTGATAA ACACCAGGGCCATGAGGGCACTAATCATAATGAGATATGCCT GCTGGAGTCGAAGTGGACCTTTCCAGTGAATGGAAAT	4213
	TGATTAGTGCCCTCATG	4214
	CATGAGGGCACTAATCA	4215
Alzheimer disease Ala231Val GCC-GTC	TTTCCATTCACTGGAAAGGTCCACTTCGACTCCAGCAGGCAT ATCTCATTATGATTAGTGCCCTCATGGCCCTGGTGTTTATCAA GTACCTCCCTGAATGGACTGCGTGGCTCATCTTGGC	4216
	GCCAAGATGAGCCACGCAGTCCATTCAGGGAGGTACTTGATA AACACCAGGGCCATGAGGGCACTAATCATAATGAGATATGCC TGCTGGAGTCGAAGTGGACCTTTCCAGTGAATGGAAA	4217
	GATTAGTGCCCTCATGG	4218
	CCATGAGGGCACTAATC	4219
Alzheimer disease Met233Thr ATG-ACG	TTCACTGGAAAGGTCCACTTCGACTCCAGCAGGCATATCTCA TTATGATTAGTGCCCTCATGGCCCTGGTGTTTATCAAGTACCT CCCTGAATGGACTGCGTGGCTCATCTTGGCTGTGAT	4220
	ATCACAGCCAAGATGAGCCACGCAGTCCATTCAGGGAGGTAC TTGATAAACACCAGGGCCATGAGGGCACTAATCATAATGAGA TATGCCTGCTGGAGTCGAAGTGGACCTTTCCAGTGAA	4221
	TGCCCTCATGGCCCTGG	4222
	CCAGGGCCATGAGGGCA	4223
Alzheimer disease Leu235Pro CTG-CCG	GGAAAGGTCCACTTCGACTCCAGCAGGCATATCTCATTATGA TTAGTGCCCTCATGGCCCTGGTGTTTATCAAGTACCTCCCTG AATGGACTGCGTGGCTCATCTTGGCTGTGATTTCAGT	4224
	ACTGAAATCACAGCCAAGATGAGCCACGCAGTCCATTCAGGG AGGTACTTGATAAACACCAGGGCCATGAGGGCACTAATCATA ATGAGATATGCCTGCTGGAGTCGAAGTGGACCTTTCC	4225
	CATGGCCCTGGTGTTTA	4226
	TAAACACCAGGGCCATG	4227
Alzheimer disease Ala246Glu GCG-GAG	TCATTATGATTAGTGCCCTCATGGCCCTGGTGTTTATCAAGTA CCTCCCTGAATGGACTGCGTGGCTCATCTTGGCTGTGATTTC AGTATATGGTAAAACCCAAGACTGATAATTTGTTTG	4228
	CAAACAAATTATCAGTCTTGGGTTTTACCATATACTGAAATCAC AGCCAAGATGAGCCACGCAGTCCATTCAGGGAGGTACTTGAT AAACACCAGGGCCATGAGGGCACTAATCATAATGA	4229
	ATGGACTGCGTGGCTCA	4230

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGAGCCACGCAGTCCAT	4231
Alzheimer disease Leu250Ser TTG-TCG	GTGCCCTCATGGCCCTGGTGTTTATCAAGTACCTCCCTGAAT GGACTGCGTGGCTCATCTTGGCTGTGATTTCAGTATATGGTA AAACCCAAGACTGATAATTTGTTTGTCACAGGAATGC	4232
	GCATTCCTGTGACAAACAAATTATCAGTCTTGGGTTTTACCAT ATACTGAAATCACAGCCAAGATGAGCCACGCAGTCCATTCAG GGAGGTACTTGATAAACACCAGGGCCATGAGGGCAC	4233
	GCTCATCTTGGCTGTGA	4234
	TCACAGCCAAGATGAGC	4235
Alzheimer disease Ala260Val GCT-GTT	AGTTTAGCCCATACATTTTATTAGATGTCTTTTATGTTTTTCTTT TTCTAGATTTAGTGGCTGTTTTGTGTCCGAAAGGTCCACTTCG TATGCTGGTTGAAACAGCTCAGGAGAGAAATGA	4236
	TCATTTCTCTCCTGAGCTGTTTCAACCAGCATACGAAGTGGAC CTTTCGGACACAAAACAGCCACTAAATCTAGAAAAAGAAAAAC ATAAAAGACATCTAATAAAATGTATGGGCTAAACT	4237
	TTTAGTGGCTGTTTTGT	4238
	ACAAAACAGCCACTAAA	4239
Alzheimer disease Leu262Phe TTGt-TTC	CCCATACATTTTATTAGATGTCTTTTATGTTTTTCTTTTTCTAGA TTTAGTGGCTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTG GTTGAAACAGCTCAGGAGAGAAATGAAACGCTT	4240
	AAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACCAGCATACGA AGTGGACCTTTCGGACACAAAACAGCCACTAAATCTAGAAAAA GAAAAACATAAAAGACATCTAATAAAATGTATGGG	4241
	GCTGTTTTGTGTCCGAA	4242
	TTCGGACACAAAACAGC	4243
Alzheimer disease Cys263Arg gTGT-CGT	CCATACATTTTATTAGATGTCTTTTATGTTTTTCTTTTTCTAGAT TTAGTGGCTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTG GTTGAAACAGCTCAGGAGAGAAATGAAACGCTTT	4244
	AAAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACCAGCATACG AAGTGGACCTTTCGGACACAAAACAGCCACTAAATCTAGAAA AAGAAAAACATAAAAGACATCTAATAAAATGTATGG	4245
	CTGTTTTGTGTCCGAAA	4246
	TTTCGGACACAAAACAG	4247
Alzheimer disease Pro264Leu CCG-CTG	ACATTTTATTAGATGTCTTTTATGTTTTTCTTTTTCTAGATTTAG TGGCTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTGGTTG AAACAGCTCAGGAGAGAAATGAAACGCTTTTTCC	4248
	GGAAAAAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACCAGCA TACGAAGTGGACCTTTCGGACACAAAACAGCCACTAAATCTA GAAAAAGAAAAACATAAAAGACATCTAATAAAATGT	4249
	TTTGTGTCCGAAAGGTC	4250
	GACCTTTCGGACACAAA	4251
Alzheimer disease Arg269Gly tCGT-GGT	GTCTTTTATGTTTTTCTTTTTCTAGATTTAGTGGCTGTTTTGTG TCCGAAAGGTCCACTTCGTATGCTGGTTGAAACAGCTCAGGA GAGAAATGAAACGCTTTTTCCAGCTCTCATTTACT	4252

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGTAAATGAGAGCTGGAAAAAGCGTTTCATTTCTCTCCTGAGC TGTTTCAACCAGCATACGAAGTGGACCTTTCGGACACAAAAC AGCCACTAAATCTAGAAAAAGAAAAACATAAAAGAC	4253
	GTCCACTTCGTATGCTG	4254
	CAGCATACGAAGTGGAC	4255
Alzheimer disease Arg269His CGT-CAT	TCTTTTATGTTTTTCTTTTTCTAGATTTAGTGGCTGTTTTGTGTC CGAAAGGTCCACTTCGTATGCTGGTTGAAACAGCTCAGGAGA GAAATGAAACGCTTTTTCCAGCTCTCATTTACTC	4256
	GAGTAAATGAGAGCTGGAAAAAGCGTTTCATTTCTCTCCTGAG CTGTTTCAACCAGCATACGAAGTGGACCTTTCGGACACAAAA CAGCCACTAAATCTAGAAAAAGAAAAACATAAAAGA	4257
	TCCACTTCGTATGCTGG	4258
	CCAGCATACGAAGTGGA	4259
Alzheimer disease Arg278Thr AGA-ACA	TAGTGGCTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTGG TTGAAACAGCTCAGGAGAGAAATGAAACGCTTTTTCCAGCTCT CATTTACTCCTGTAAGTATTTGAGAATGATATTGAA	4260
	TTCAATATCATTCTCAAATACTTACAGGAGTAAATGAGAGCTG GAAAAAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACCAGCAT ACGAAGTGGACCTTTCGGACACAAAACAGCCACTA	4261
	TCAGGAGAGAAATGAAA	4262
	TTTCATTTCTCTCCTGA	4263
Alzheimer disease Glu280Ala GAA-GCA	CTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTGGTTGAAAC AGCTCAGGAGAGAAATGAAAACGCTTTTTCCAGCTCTCATTTAC TCCTGTAAGTATTTGAGAATGATATTGAATTAGTA	4264
	TACTAATTCAATATCATTCTCAAATACTTACAGGAGTAAATGAG AGCTGGAAAAAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACC AGCATACGAAGTGGACCTTTCGGACACAAAACAG	4265
	GAGAAATGAAAACGCTTT	4266
	AAAGCGTTTCATTTCTC	4267
Alzheimer disease Glu280Gly GAA-GGA	CTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTGGTTGAAAC AGCTCAGGAGAGAAATGAAAACGCTTTTTCCAGCTCTCATTTAC TCCTGTAAGTATTTGAGAATGATATTGAATTAGTA	4268
	TACTAATTCAATATCATTCTCAAATACTTACAGGAGTAAATGAG AGCTGGAAAAAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACC AGCATACGAAGTGGACCTTTCGGACACAAAACAG	4269
	GAGAAATGAAAACGCTTT	4270
	AAAGCGTTTCATTTCTC	4271
Alzheimer disease Leu282Arg CTT-CGT	TGTGTCCGAAAGGTCCACTTCGTATGCTGGTTGAAACAGCTC AGGAGAGAAATGAAACGCTTTTTCCAGCTCTCATTTACTCCTG TAAGTATTTGAGAATGATATTGAATTAGTAATCAGT	4272
	ACTGATTACTAATTCAATATCATTCTCAAATACTTACAGGAGTA AATGAGAGCTGGAAAAAGCGTTTCATTTCTCTCCTGAGCTGTT TCAACCAGCATACGAAGTGGACCTTTCGGACACA	4273
	TGAAACGCTTTTTCCAG	4274

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTGGAAAAAGCGTTTCA	4275
Alzheimer disease Ala285Val GCT-GTT	AAGGTCCACTTCGTATGCTGGTTGAAACAGCTCAGGAGAGAA ATGAAACGCTTTTTCCAGCTCTCATTTACTCCTGTAAGTATTTG AGAATGATATTGAATTAGTAATCAGTGTAGAATTT	4276
	AAATTCTACACTGATTACTAATTCAATATCATTCTCAAATACTTA CAGGAGTAAATGAGAGCTGGAAAAAGCGTTTCATTTCTCTCCT GAGCTGTTTCAACCAGCATACGAAGTGGACCTT	4277
	TTTTCCAGCTCTCATTT	4278
	AAATGAGAGCTGGAAAA	4279
Alzheimer disease Leu286Val tCTC-GTC	GGTCCACTTCGTATGCTGGTTGAAACAGCTCAGGAGAGAAAT GAAACGCTTTTTCCAGCTCTCATTTACTCCTGTAAGTATTTGA GAATGATATTGAATTAGTAATCAGTGTAGAATTTAT	4280
	ATAAATTCTACACTGATTACTAATTCAATATCATTCTCAAATACT TACAGGAGTAAATGAGAGCTGGAAAAAGCGTTTCATTTCTCTC CTGAGCTGTTTCAACCAGCATACGAAGTGGACC	4281
	TTCCAGCTCTCATTTAC	4282
	GTAAATGAGAGCTGGAA	4283
Alzheimer disease Gly384Ala GGA-GCA	GTGACCAACTTTTTAATATTTGTAACCTTTCCTTTTTAGGGGGA GTAAAACTTGGATTGGGAGATTTCATTTTCTACAGTGTTCTGG TTGGTAAAGCCTCAGCAACAGCCAGTGGAGACTG	4284
	CAGTCTCCACTGGCTGTTGCTGAGGCTTTACCAACCAGAACA CTGTAGAAAATGAAATCTCCCAATCCAAGTTTTACTCCCCCTA AAAAGGAAAGGTTACAAATATTAAAAAGTTGGTCAC	4285
	TGGATTGGGAGATTTCA	4286
	TGAAATCTCCCAATCCA	4287
Alzheimer disease Ser390Ile AGT-ATT	TTTGTAACCTTTCCTTTTTAGGGGGAGTAAAACTTGGATTGGG AGATTTCATTTTCTACAGTGTTCTGGTTGGTAAAGCCTCAGCA ACAGCCAGTGGAGACTGGAACACAACCATAGCCTG	4288
	CAGGCTATGGTTGTGTTCCAGTCTCCACTGGCTGTTGCTGAG GCTTTACCAACCAGAACACTGTAGAAAATGAAATCTCCCAATC CAAGTTTTACTCCCCCTAAAAAGGAAAGGTTACAAA	4289
	TTTCTACAGTGTTCTGG	4290
	CCAGAACACTGTAGAAA	4291
Alzheimer disease Leu392Val tCTG-GTG	AACCTTTCCTTTTTAGGGGGAGTAAAACTTGGATTGGGAGATT TCATTTTCTACAGTGTTCTGGTTGGTAAAGCCTCAGCAACAGC CAGTGGAGACTGGAACACAACCATAGCCTGTTTCG	4292
	CGAAACAGGCTATGGTTGTGTTCCAGTCTCCACTGGCTGTTG CTGAGGCTTTACCAACCAGAACACTGTAGAAAATGAAATCTCC CAATCCAAGTTTTACTCCCCCTAAAAAGGAAAGGTT	4293
	ACAGTGTTCTGGTTGGT	4294
	ACCAACCAGAACACTGT	4295
Alzheimer disease Asn405Ser AAC-AGC	ATTTCATTTTCTACAGTGTTCTGGTTGGTAAAGCCTCAGCAAC AGCCAGTGGAGACTGGAACACAACCATAGCCTGTTTCGTAGC CATATTAATTGTAAGTATACACTAATAAGAATGTGT	4296

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACACATTCTTATTAGTGTATACTTACAATTAATATGGCTACGAA ACAGGCTATGGTTGTGTTCCAGTCTCCACTGGCTGTTGCTGA GGCTTTACCAACCAGAACACTGTAGAAAATGAAAT	4297
	AGACTGGAACACAACCA	4298
	TGGTTGTGTTCCAGTCT	4299
Alzheimer disease Ala409Thr aGCC-ACC	TACAGTGTTCTGGTTGGTAAAGCCTCAGCAACAGCCAGTGGA GACTGGAACACAACCATAGCCTGTTTCGTAGCCATATTAATTG TAAGTATACACTAATAAGAATGTGTCAGAGCTCTTA	4300
	TAAGAGCTCTGACACATTCTTATTAGTGTATACTTACAATTAAT ATGGCTACGAAACAGGCTATGGTTGTGTTCCAGTCTCCACTG GCTGTTGCTGAGGCTTTACCAACCAGAACACTGTA	4301
	CAACCATAGCCTGTTTC	4302
	GAAACAGGCTATGGTTG	4303
Alzheimer disease Cys410Tyr TGT-TAT	GTGTTCTGGTTGGTAAAGCCTCAGCAACAGCCAGTGGAGACT GGAACACAACCATAGCCTGTTTCGTAGCCATATTAATTGTAAG TATACACTAATAAGAATGTGTCAGAGCTCTTAATGT	4304
	ACATTAAGAGCTCTGACACATTCTTATTAGTGTATACTTACAAT TAATATGGCTACGAAACAGGCTATGGTTGTGTTCCAGTCTCCA CTGGCTGTTGCTGAGGCTTTACCAACCAGAACAC	4305
	CATAGCCTGTTTCGTAG	4306
	CTACGAAACAGGCTATG	4307
Alzheimer disease Ala426Pro tGCC-CCC	TGTGAATGTGTGTCTTTCCCATCTTCTCCACAGGGTTTGTGCC TTACATTATTACTCCTTGCCATTTTCAAGAAAGCATTGCCAGCT CTTCCAATCTCCATCACCTTTGGGCTTGTTTTCT	4308
	AGAAAACAAGCCCAAAGGTGATGGAGATTGGAAGAGCTGGCA ATGCTTTCTTGAAAATGGCAAGGAGTAATAATGTAAGGCACAA ACCCTGTGGAGAAGATGGGAAAGACACACATTCACA	4309
	TACTCCTTGCCATTTTC	4310
	GAAAATGGCAAGGAGTA	4311
Alzheimer disease Pro436Gln CCA-CAA	AGGGTTTGTGCCTTACATTATTACTCCTTGCCATTTTCAAGAA AGCATTGCCAGCTCTTCCAATCTCCATCACCTTTGGGCTTGTT TTCTACTTTGCCACAGATTATCTTGTACAGCCTTT	4312
	AAAGGCTGTACAAGATAATCTGTGGCAAAGTAGAAAACAAGC CCAAAGGTGATGGAGATTGGAAGAGCTGGCAATGCTTTCTTG AAAATGGCAAGGAGTAATAATGTAAGGCACAAACCCT	4313
	AGCTCTTCCAATCTCCA	4314
	TGGAGATTGGAAGAGCT	4315
Alzheimer disease Pro436Ser tCCA-TCA	CAGGGTTTGTGCCTTACATTATTACTCCTTGCCATTTTCAAGA AAGCATTGCCAGCTCTTCCAATCTCCATCACCTTTGGGCTTGT TTTCTACTTTGCCACAGATTATCTTGTACAGCCTT	4316
	AAGGCTGTACAAGATAATCTGTGGCAAAGTAGAAAACAAGCC CAAAGGTGATGGAGATTGGAAGAGCTGGCAATGCTTTCTTGA AAATGGCAAGGAGTAATAATGTAAGGCACAAACCCTG	4317
	CAGCTCTTCCAATCTCC	4318

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GGAGATTGGAAGAGCTG	4319

EXAMPLE 25
Alzheimer's Disease - presenilin-2 (PSEN2)

The attached table discloses the correcting oligonucleotide base sequences for the PSEN2 oligonucleotides of the invention.

Table 32
PSEN2 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Arg62His CGC-CAC	GATGTGGTTTCCCACAGAGAAGCCAGGAGAACGAGGAGGAC GGTGAGGAGGACCCTGACCGCTATGTCTGTAGTGGGGTTCC CGGGCGGCCGCCAGGCCTGGAGGAAGAGCTGACCCTCAA	4320
	TTGAGGGTCAGCTCTTCCTCCAGGCCTGGCGGCCGCCCGGG AACCCCACTACAGACATAGCGGTCAGGGTCCTCCTCACCGTC CTCCTCGTTCTCCTGGCTTCTCTGTGGGAAACCACATC	4321
	CCCTGACCGCTATGTCT	4322
	AGACATAGCGGTCAGGG	4323
Alzheimer disease Thr122Pro cACG-CCG	GCCTCGAGGAGCAGTCAGGGCCGGGAGCATCAGCCCTTTGC CTTCTCCCTCAGCATCTACACGACATTCACTGAGGACACACC CTCGGTGGGCCAGCGCCTCCTCAACTCCGTGCTGAACA	4324
	TGTTCAGCACGGAGTTGAGGAGGCGCTGGCCCACCGAGGGT GTGTCCTCAGTGAATGTCGTGTAGATGCTGAGGGAGAAGGCA AAGGGCTGATGCTCCCGGCCCTGACTGCTCCTCGAGGC	4325
	GCATCTACACGACATTC	4326
	GAATGTCGTGTAGATGC	4327
Alzheimer disease Asn141Ile AAC-ATC	ACACGCCATTCACTGAGGACACACCCTCGGTGGGCCAGCGC CTCCTCAACTCCGTGCTGAACACCCTCATCATGATCAGCGTC ATCGTGGTTATGACCATCTTCTTGGTGGTGCTCTACAA	4328
	TTGTAGAGCACCACCAAGAAGATGGTCATAACCACGATGACG CTGATCATGATGAGGGTGTTCAGCACGGAGTTGAGGAGGCG CTGGCCCACCGAGGGTGTGTCCTCAGTGAATGGCGTGT	4329
	CGTGCTGAACACCCTCA	4330
	TGAGGGTGTTCAGCACG	4331
Alzheimer disease Met239Ile ATGg-ATA	CCACTGGAAGGGCCCTCTGGTGCTGCAGCAGGCCTACCTCA TCATGATCAGTGCGCTCATGGCCCTAGTGTTCATCAAGTACCT CCCAGAGTGGTCCGCGTGGGTCATCCTGGGCGCCATC	4332

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GATGGCGCCCAGGATGACCCACGCGGACCACTCTGGGAGGT ACTTGATGAACACTAGGGC**C**ATGAGCGCACTGATCATGATGA GGTAGGCCTGCTGCAGCACCAGAGGGCCCTTCCAGTGG	4333
	GCGCTCAT**G**GCCCTAGT	4334
	ACTAGGGC**C**ATGAGCGC	4335
Alzheimer disease Met239Val cATG-GTG	ATCCACTGGAAGGGCCCTCTGGTGCTGCAGCAGGCCTACCT CATCATGATCAGTGCGCTC**A**TGGCCCTAGTGTTCATCAAGTA CCTCCCAGAGTGGTCCGCGTGGGTCATCCTGGGCGCCA	4336
	TGGCGCCCAGGATGACCCACGCGGACCACTCTGGGAGGTAC TTGATGAACACTAGGGCCA**T**GAGCGCACTGATCATGATGAGG TAGGCCTGCTGCAGCACCAGAGGGCCCTTCCAGTGGAT	4337
	GTGCGCTC**A**TGGCCCTA	4338
	TAGGGCCA**T**GAGCGCAC	4339

EXAMPLE 26
Plant Cells

The oligonucleotides of the invention can also be used to repair or direct a mutagenic event in plants and animal cells. Although little information is available on plant mutations amongst natural cultivars, the oligonucleotides of the invention can be used to produce "knock out" mutations by modification of specific amino acid codons to produce stop codons (e.g., a CAA codon specifying Gln can be modified at a specific site to TAA; a AAG codon specifying Lys can be modified to UAG at a specific site; and a CGA codon for Arg can be modified to a UGA codon at a specific site). Such base pair changes will terminate the reading frame and produce a defective truncated protein, shortened at the site of the stop codon. Alternatively, frameshift additions or deletions can be directed into the genome at a specific sequence to interrupt the reading frame and produce a garbled downstream protein. Such stop or frameshift mutations can be introduced to determine the effect of knocking out the protein in either plant or animal cells.

All publications and patent applications cited in this specification are herein incorporated by reference as if each individual publication or patent application were specifically and individually indicated to be incorporated by reference. Although the foregoing invention has been described in some detail by way of illustration and example for purposes of clarity of understanding, it will be readily apparent to those of ordinary skill in the art in light of the teachings of this invention that certain changes and modifications may be made thereto without departing from the spirit or scope of the appended claims.